10/000,039

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     (FILE 'HOME' ENTERED AT 09:30:06 ON 28 MAY 2004)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS'
     ENTERED AT 09:30:37 ON 28 MAY 2004
L1           881 S VOLUME(A)REGULATED
L2           157 S L1 AND KINASE?
L3            49 S HUMAN AND L2
L4            19 DUP REM L3 (30 DUPLICATES REMOVED)
L5            39 S "H-SGK"
L6            20 DUP REM L5 (19 DUPLICATES REMOVED)
L7            20 S L6 AND KINASE?
L8       6092312 S CLON? OR EXPRESS? OR RECOMBINANT
L9           73 S L3 OR L5
L10          49 S L9 AND L8
L11          22 DUP REM L10 (27 DUPLICATES REMOVED)
                E LANG F/AU
L12        3108 S E3
                E WALDEGGER S/AU
L13         348 S E3
L14        3155 S L12 OR L13
L15          30 S L9 AND L14
L16          15 DUP REM L15 (15 DUPLICATES REMOVED)

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 NEWS  3  JAN 27  Source of Registration (SR) information in REGISTRY updated
                  and searchable
 NEWS  4  JAN 27  A new search aid, the Company Name Thesaurus, available in
                  CA/CAplus
 NEWS  5  FEB 05  German (DE) application and patent publication number format
                  changes
 NEWS  6  MAR 03  MEDLINE and LMEDLINE reloaded
 NEWS  7  MAR 03  MEDLINE file segment of TOXCENTER reloaded
 NEWS  8  MAR 03  FRANCEPAT now available on STN
 NEWS  9  MAR 29  Pharmaceutical Substances (PS) now available on STN
 NEWS 10  MAR 29  WPIFV now available on STN
 NEWS 11  MAR 29  New monthly current-awareness alert (SDI) frequency in RAPRA
 NEWS 12  APR 26  PROMT: New display field available
 NEWS 13  APR 26  IFIPAT/IFIUDB/IFICDB: New super search and display field
                  available
 NEWS 14  APR 26  LITALERT now available on STN
 NEWS 15  APR 27  NLDB: New search and display fields available
 NEWS 16  May 10  PROUSDDR now available on STN
 NEWS 17  May 19  PROUSDDR: One FREE connect hour, per account, in both May
                  and June 2004
 NEWS 18  May 12  EXTEND option available in structure searching
 NEWS 19  May 12  Polymer links for the POLYLINK command completed in REGISTRY
 NEWS 20  May 17  FRFULL now available on STN
 NEWS 21  May 27  STN User Update to be held June 7 and June 8 at the SLA 2004
                  Conference
 NEWS 22  May 27  New UPM (Update Code Maximum) field for more efficient patent
                  SDIs in CAplus
 NEWS 23  May 27  CAplus super roles and document types searchable in REGISTRY
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               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 26 APRIL 2004
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FILE 'HOME' ENTERED AT 09:30:06 ON 28 MAY 2004

=> file medline embase biosis biotechds scisearch hcaplus ntis
COST IN U.S. DOLLARS

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FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 09:30:37 ON 28 MAY 2004

FILE 'EMBASE' ENTERED AT 09:30:37 ON 28 MAY 2004

FILE 'BIOSIS' ENTERED AT 09:30:37 ON 28 MAY 2004

FILE 'BIOTECHDS' ENTERED AT 09:30:37 ON 28 MAY 2004

FILE 'SCISEARCH' ENTERED AT 09:30:37 ON 28 MAY 2004

FILE 'HCAPLUS' ENTERED AT 09:30:37 ON 28 MAY 2004

FILE 'NTIS' ENTERED AT 09:30:37 ON 28 MAY 2004

=> s volume(a)regulated
L1 881 VOLUME(A) REGULATED

=> s l1 and kinase?
L2 157 L1 AND KINASE?

=> s human and l2
L3 49 HUMAN AND L2

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 19 DUP REM L3 (30 DUPLICATES REMOVED)

=> d 1-19 ibib ab

L4 ANSWER 1 OF 19 MEDLINE on STN
ACCESSION NUMBER: 2004252865 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 14960416
TITLE: Osmotic swelling-provoked release of organic osmolytes in **human** intestinal epithelial cells.
AUTHOR: Tomassen Sebastian F B; Fekkes Durk; de Jonge Hugo R; Tilly Ben C
CORPORATE SOURCE: Department of Biochemistry, Erasmus University Medical Center, 3000 DR Rotterdam, The Netherlands.
SOURCE: American journal of physiology. Cell physiology, (2004 Jun) 286 (6) C1417-22.
Journal code: 100901225. ISSN: 0363-6143.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20040521
Last Updated on STN: 20040525

AB **Human** Intestine 407 cells respond to osmotic cell swelling by the activation of Cl(-)- and K(+)-selective ionic channels, as well as by stimulating an organic osmolyte release pathway readily permeable to taurine and phosphocholine. Unlike the activation of **volume-regulated** anion channels (VRAC), activation of the organic osmolyte release pathway shows a lag time of approximately 30-60 s, and its activity persists for at least 8-12 min. In contrast to VRAC activation, stimulation of organic osmolyte release did not require protein tyrosine phosphorylation, active p21(rho), or phosphatidylinositol 3-**kinase** activity and was insensitive to Cl(-) channel blockers. Treatment of the cells with putative organic anion transporter inhibitors reduced the release of taurine only partially or was found to be ineffective. The efflux was blocked by a subclass of organic cation transporter (OCT) inhibitors (cyanine-863 and decynium-22) but not by other OCT inhibitors (cimetidine, quinine, and verapamil). Brief treatment of the cells with phorbol esters potentiated the cell swelling-induced taurine efflux, whereas addition of the protein **kinase** C (PKC) inhibitor GF109203X largely inhibited the response, suggesting that PKC is involved. Increasing the level of intracellular Ca(2+) by using A-23187- or Ca(2+)-mobilizing hormones, however, did not affect the magnitude of the response. Taken together, the results indicate that the hypotonicity-induced efflux of organic osmolytes is independent of VRAC and involves a PKC-dependent step.

L4 ANSWER 2 OF 19 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN DUPLICATE 1
ACCESSION NUMBER: 2003249750 EMBASE
TITLE: Store-operated Ca(2+) channels in prostate cancer epithelial cells: Function, regulation, and role in carcinogenesis.
AUTHOR: Vanden Abeele F.; Shuba Y.; Roudbaraki M.; Lemonnier L.; Vanoverberghe K.; Mariot P.; Skryma R.; Prevarskaya N.
CORPORATE SOURCE: N. Prevarskaya, Laboratoire Physiologie Cellulaire, INSERM EMI 0228, USTL, 59655 Villeneuve d'Ascq, France. Natacha.Prevarskaya@univ-lille1.fr
SOURCE: Cell Calcium, (2003) 33/5 (357-373).
Refs: 77
ISSN: 0143-4160 CODEN: CECADV
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
016 Cancer
022 Human Genetics
028 Urology and Nephrology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Ca(2+) homeostasis mechanisms, in which the Ca(2+) entry pathways play a key role, are critically involved in both normal function and cancerous transformation of prostate epithelial cells. Here, using the lymph node carcinoma of the prostate (LNCaP) cell line as a major experimental model, we characterize prostate-specific store-operated Ca(2+) channels (SOCs) - a primary Ca(2+) entry pathway for non-excitable cells - for the first time. We show that prostate-specific SOCs share major store-dependent, kinetic, permeation, inwardly rectifying, and pharmacological (including dual, potentiation/inhibition concentration-dependent sensitivity to 2-APB) properties with "classical" Ca(2+) release-activated Ca(2+) channels (CRAC), but have a higher single channel conductance (3.2 and 12pS in Ca(2+)- and Na(+) -permeable modes, respectively). They are subject to feedback inhibition via Ca(2+) -dependent PKC, CaMK-II and CaM

regulatory pathways and are functionally dependent on caveolae integrity. Caveolae also provide a scaffold for spatial co-localization of SOCs with **volume-regulated** anion channels (VRAC) and their Ca(2+) -mediated interaction. The TRPC1 and TRPV6 members of the transient receptor potential (TRP) channel family are the most likely molecular candidates for the formation of prostate-specific endogenous SOCs. Differentiation of LNCaP cells to an androgen-insensitive, apoptotic-resistant neuroendocrine phenotype downregulates SOC current. We conclude that prostate-specific SOCs are important determinants in the transition to androgen-independent prostate cancer.

L4 ANSWER 3 OF 19 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2002328933 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11976941
TITLE: Myosin light chain **kinase** modulates hypotonicity-induced Ca2+ entry and Cl- channel activity in **human** cervical cancer cells.
AUTHOR: Shen Meng-Ru; Furla Paola; Chou Cheng-Yang; Ellory J Clive
CORPORATE SOURCE: Department of Obstetrics and Gynaecology, College of Medicine, National Cheng Kung University, Tainan 704, Taiwan.
SOURCE: Pflugers Archiv : European journal of physiology, (2002 May) 444 (1-2) 276-85.
Journal code: 0154720. ISSN: 0031-6768.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200211
ENTRY DATE: Entered STN: 20020620
Last Updated on STN: 20030105
Entered Medline: 20021104

AB Hypotonicity-induced Ca2+ entry is a critical signal for the normal regulatory volume decrease in **human** cervical cancer cells. The aim of this study was to explore the role of myosin light chain **kinase** (MLCK) in the regulation of hypotonicity-induced Ca2+ signalling and Cl- channel activity. Blockade of MLCK activity by MLCK(11-19) amide, a substrate-specific peptide inhibitor, markedly attenuated hypotonicity-induced Ca2+ entry. A similar result was obtained with ML-7, a synthetic naphthalenesulphonyl derivative that inhibits the binding of ATP to MLCK. More than 85% of the activity of the **volume-regulated** Cl- channel was suppressed when intracellular Ca2+ was buffered to near zero in the absence of extracellular Ca2+, suggesting that hypotonicity-induced Ca2+ signalling is important for the activation of the **volume-regulated** Cl- channel. Intracellular dialysis with MLCK(11-19) amide or ML-7 concentration-dependently reduced the amplitude and rate of activation of the **volume-regulated** Cl- channel. Swelling-activated taurine transport was also inhibited concentration dependently by ML-7 and MLCK(11-19) amide with IC(50) values of 6.4 and 2.0 microM, respectively. Hypotonicity induced MLC phosphorylation which was mediated totally by MLCK and depended on Ca2+ entry. However, phosphorylated MLC per se was not involved critically in the regulation of Ca2+ entry and activation of volume-sensitive organic osmolyte/anion channels (VSOAC). We propose that MLCK has a novel function in regulating the activation of VSOAC by mediating Ca2+ entry in response to hypotonicity. This function of MLCK on Ca2+ signalling does not correlate with MLC phosphorylation.

L4 ANSWER 4 OF 19 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2002312565 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12055079
TITLE: RhoA exerts a permissive effect on **volume-regulated** anion channels in vascular endothelial

cells.
AUTHOR: Carton Iris; Trouet Dominique; Hermans Diane; Barth Holger; Aktories Klaus; Droogmans Guy; Jorgensen Nanna K; Hoffmann Else K; Nilius Bernd; Eggermont Jan
CORPORATE SOURCE: Laboratory of Physiology, Katholieke Universiteit Leuven, Campus Gasthuisberg, B-3000 Leuven, Belgium.
SOURCE: American journal of physiology. Cell physiology, (2002 Jul) 283 (1) C115-25.
Journal code: 100901225. ISSN: 0363-6143.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200207
ENTRY DATE: Entered STN: 20020611
Last Updated on STN: 20020717
Entered Medline: 20020716

AB Cell swelling triggers in most cell types an outwardly rectifying anion current, I(Cl,swell), via **volume-regulated** anion channels (VRACs). We have previously demonstrated in calf pulmonary artery endothelial (CPAE) cells that inhibition of the Rho/Rho **kinase**/myosin light chain phosphorylation pathway reduces the swelling-dependent activation of I(Cl,swell). However, these experiments did not allow us to discriminate between a direct activator role or a permissive effect. We now show that the Rho pathway did not affect VRAC activity if this pathway was activated by transfecting CPAE cells with constitutively active isoforms of Galpha (a Rho activating heterotrimeric G protein subunit), Rho, or Rho **kinase**. Furthermore, biochemical and morphological analysis failed to demonstrate activation of the Rho pathway during hypotonic cell swelling. Finally, manipulating the Rho pathway with either guanosine 5'-O-(3-thiotriphosphate) or C3 exoenzyme had no effect on VRACs in caveolin-1-expressing Caco-2 cells. We conclude that the Rho pathway exerts a permissive effect on VRACs in CPAE cells, i.e., swelling-induced opening of VRACs requires a functional Rho pathway, but not an activation of the Rho pathway.

L4 ANSWER 5 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:74113 BIOSIS
DOCUMENT NUMBER: PREV200200074113
TITLE: Cell **volume-regulated human kinase** h-sgk.
AUTHOR(S): Lang, Florian [Inventor, Reprint author]; Waldegger, Siegfried [Inventor]
CORPORATE SOURCE: Im Rotbad 52, 72076 Tubingen, Germany
PATENT INFORMATION: US 6326181 December 04, 2001
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Dec. 4, 2001) Vol. 1253, No. 1.
ftp://ftp.uspto.gov/pub/patdata/. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jan 2002
Last Updated on STN: 25 Feb 2002

AB The present invention relates to the cloning and characterization of a **human** serine/threonine **kinase** (h-sgk: serum and glucocorticoid dependent **kinase**). The invention furthermore relates to reagents for diagnosing conditions associated with a change in cell volume and/or in "macromolecular crowding" in the body, such as, for example, hypernatremia, hyponatremia, diabetes mellitus, renal failure, hypercatabolism, hepatic encephalopathy, inflammation and microbial or viral infections. The present invention additionally relates to pharmaceuticals comprising the h-sgk, nucleic acids which code for the h-sgk, or receptors, in particular antibodies, which specifically bind to the h-sgk.

L4 ANSWER 6 OF 19 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2002179776 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11913450
TITLE: Serum- and glucocorticoid-dependent **kinase**, cell volume, and the regulation of epithelial transport.
AUTHOR: Fillon S; Warntges S; Matskevitch J; Moschen I; Setiawan I; Gamper N; Feng Y X; Stegen C; Friedrich B; Waldegger S; Broer S; Wagner C A; Huber S M; Klingel K; Vereninov A; Lang F
CORPORATE SOURCE: Department of Physiology, University of Tubingen, Germany.
SOURCE: Comparative biochemistry and physiology. Part A, Molecular & integrative physiology, (2001 Oct) 130 (3) 367-76. Ref: 99
Journal code: 9806096. ISSN: 1095-6433.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20020401
Last Updated on STN: 20020614
Entered Medline: 20020418

AB Ample pharmacological evidence points to a role of **kinases** in the regulation of cell volume. Given the limited selectivity of most inhibitors, however, the specific molecules involved have remained largely elusive. The search for cell **volume regulated** genes in liver HepG2 cells led to the discovery of the **human** serum- and glucocorticoid-dependent serine/threonine **kinase** hsgk1. Transcription and expression of hsgk1 is markedly and rapidly upregulated by osmotic and isotonic cell shrinkage. The effect of osmotic cell shrinkage on hsgk1 is mediated by p38 **kinase**. Further stimuli of hsgk1 transcription include glucocorticoids, aldosterone, TGF-beta1, serum, increase of intracellular Ca2+ and phorbolesters, whereas cAMP downregulates hsgk1 transcription. The hsgk1 protein is expressed in several epithelial tissues including **human** pancreas, intestine, kidney, and shark rectal gland. Co-expression of hsgk1 with the renal epithelial Na+-channel ENaC or the Na+/K+/2Cl(-)-cotransporter NKCC2 (BSC1) in Xenopus oocytes, accelerates insertion of the transport proteins into the cell membrane and thus, stimulates channel or transport activity. Thus, hsgk1 participates in the regulation of transport by steroids and secretagogues increasing intracellular Ca2+-activity. The stimulation of hsgk1 transcription by TGF-beta1 may further bear pathophysiological relevance.

L4 ANSWER 7 OF 19 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2002:18079 SCISEARCH
THE GENUINE ARTICLE: 506HT
TITLE: Ca2+ modulation of **volume-regulated** anion channels: evidence for colocalization with store-operated channels
AUTHOR: Lemonnier L; Prevarskaya N; Shuba Y; Vanden Abeele F; Nilius B; Mazurier J; Skryma R (Reprint)
CORPORATE SOURCE: Univ Sci & Technol Lille, Lab Physiol Cellulaire, INSERM, EPI 9938, Batiment SN3, F-59655 Villeneuve Dascq, France (Reprint); Univ Sci & Technol Lille, Lab Physiol Cellulaire, INSERM, EPI 9938, F-59655 Villeneuve Dascq, France; Bogomoletz Inst Physiol, UA-01024 Kiev 24, Ukraine; Katholieke Univ Leuven, Fysiol Lab, B-3000 Louvain, Belgium
COUNTRY OF AUTHOR: France; Ukraine; Belgium
SOURCE: FASEB JOURNAL, (DEC 2001) Vol. 15, No. 14, pp. U297-U314.

Publisher: FEDERATION AMER SOC EXP BIOL, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998 USA.
ISSN: 0892-6638.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 50
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Ca2+ regulation of Cl- current induced by cell swelling (I-Cl,I-swell) in response to hypotonicity was studied in **human** prostate cancer epithelial cells (LNCaP) by using the patch-clamp technique. Increase of global intracellular Ca2+ ([Ca2+](in)) to 1 muM as well as variations of the extracellular Ca2+ ([Ca2+](out)) in the 0 to 10 mM range did not affect time course of the development, maximal amplitude, rectification properties, and kinetics of I-Cl,I-swell. However, the presence of 0.1 muM thapsigargin (TG), an inhibitor of endoplasmic reticulum (ER) Ca2+ pump, resulted in a more than 50% inhibition of I-Cl,I-swell. The blockade of plasma membrane store-operated channels (SOCs), activated in the presence of TG, by 2 mM Ni2+ prevented TG-conferred I-Cl,I-swell inhibition by extracellular Ca2+. In the presence of TG and Ca2+, the cells failed to exhibit regulatory volume decrease. We conclude that interaction between **volume-regulated** anion channels (VRACs) carrying I-Cl,I-swell and Ca2+ occurs in the microdomains from the inner surface of the membrane that are not accessible to the changes in [Ca2+](in), but can be readily reached by Ca2+ entering the cell via plasma membrane, especially through SOCs. Preferred access of SOC-transported Ca2+ to VRAC suggests colocalization of these channels in the cell membrane.

L4 ANSWER 8 OF 19 MEDLINE on STN
ACCESSION NUMBER: 2002163457 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11894846
TITLE: Cellular function and control of **volume-regulated** anion channels.
AUTHOR: Eggermont J; Trouet D; Carton I; Nilius B
CORPORATE SOURCE: Laboratory of Physiology, Catholic University of Leuven, Belgium.. Jan.Eggermont@med.kuleuven.ac.be
SOURCE: Cell biochemistry and biophysics, (2001) 35 (3) 263-74. Ref: 82
Journal code: 9701934. ISSN: 1085-9195.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200208
ENTRY DATE: Entered STN: 20020317
Last Updated on STN: 20020827
Entered Medline: 20020826

AB Restoration of cell volume after cell swelling in mammalian cells is achieved by the loss of solutes (K+, Cl-, and organic osmolytes) and the subsequent osmotically driven efflux of water. This process is generally known as regulatory volume decrease (RVD). One pathway for the swelling induced loss of Cl- (and also organic osmolytes) during RVD is the **volume-regulated** anion channel (VRAC). In this review, we discuss the physiological role and cellular control of VRAC. We will first highlight evidence that VRAC is more than a volume regulator and that it participates in other fundamental cellular processes such as cell proliferation and apoptosis. The second part concentrates on the Rho/Rho **kinase**/myosin phosphorylation cascade and on compartmentalization in caveolae as modulators of the signal transduction cascade that controls VRAC gating in vascular endothelial cells.

L4 ANSWER 9 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:756527 HCAPLUS

DOCUMENT NUMBER: 133:325643
TITLE: Antifibrotic formulations containing inhibitors of cell-**volume-regulated** **human kinase** h-sgk
INVENTOR(S): Lang, Florian; Waldegger, Siegfried; Wagner, Carsten; Broer, Stefan; Klingel, Karin
PATENT ASSIGNEE(S): Germany
SOURCE: PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000062781	A1	20001026	WO 2000-EP3578	20000419
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
DE 19917990	A1	20001102	DE 1999-19917990	19990420
BR 2000009914	A	20020108	BR 2000-9914	20000419
EP 1171131	A1	20020116	EP 2000-922655	20000419
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002542196	T2	20021210	JP 2000-611917	20000419
NO 2001005054	A	20011214	NO 2001-5054	20011017
ZA 2001008610	A	20020102	ZA 2001-8610	20011019

PRIORITY APPLN. INFO.: DE 1999-19917990 A 19990420
WO 2000-EP3578 W 20000419

AB The invention relates to medicaments which contain inhibitors or activators of cell-**vol.-regulated human** serum and glucocorticoid-dependent **kinase** h-sgk, a serine-threonine **kinase**. Medicaments of this type containing staurosporin or chelerythrine are suitable for treating conditions, such as fibrosis, in which an increased or reduced expression of h-sgk is identified.

REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 10 OF 19 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 2001013129 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11003595
TITLE: **Volume-regulated** chloride conductance in the LNCaP **human** prostate cancer cell line.
AUTHOR: Shuba Y M; Prevarskaya N; Lemonnier L; Van Coppenolle F; Kostyuk P G; Mauroy B; Skryma R
CORPORATE SOURCE: Laboratoire de Physiologie Cellulaire, Institut National de la Sante et de la Recherche Medicale EPI 9938, Universite des Sciences et Technologies de Lille, 59655 Villeneuve d'Ascq, France.. phycel@pop.univ-lille1.fr
SOURCE: American journal of physiology. Cell physiology, (2000 Oct) 279 (4) C1144-54.
Journal code: 100901225. ISSN: 0363-6143.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001030

AB Patch-clamp recordings were used to study ion currents induced by cell swelling caused by hypotonicity in **human** prostate cancer epithelial cells, LNCaP. The reversal potential of the swelling-evoked current suggested that Cl(-) was the primary charge carrier (termed I(Cl,swell)). The selectivity sequence of the underlying **volume -regulated** anion channels (VRACs) for different anions was Br(-) approximately I(-) > Cl(-) > F(-) > methanesulfonate >> glutamate, with relative permeability numbers of 1.26, 1.20, 1.0, 0.77, 0.49, and 0.036, respectively. The current-voltage patterns of the whole cell currents as well as single-channel currents showed moderate outward rectification. Unitary VRAC conductance was determined at 9.6 +/- 1.8 pS. Conventional Cl(-) channel blockers 5-nitro-2-(3-phenylpropylamino)benzoic acid (100 microM) and DIDS (100 microM) inhibited whole cell I(Cl,swell) in a voltage-dependent manner, with the block decreasing from 39.6 +/- 9.7% and 71.0 +/- 11. 0% at +50 mV to 26.2 +/- 7.2% and 14.5 +/- 6.6% at -100 mV, respectively. Verapamil (50 microM), a standard Ca(2+) antagonist and P-glycoprotein function inhibitor, depressed the current by a maximum of 15%. Protein tyrosine **kinase** inhibitors downregulated I(Cl,swell) (genistein with an IC(50) of 2.6 microM and lavendustin A by 60 +/- 14% at 1 microM). The protein tyrosine phosphatase inhibitor sodium orthovanadate (500 microM) stimulated I(Cl,swell) by 54 +/- 11%. We conclude that VRACs in **human** prostate cancer epithelial cells are modulated via protein tyrosine phosphorylation.

L4 ANSWER 11 OF 19 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 2001034894 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11052997
TITLE: Expression of cell **volume-regulated kinase** h-sgk in pancreatic tissue.
AUTHOR: Klingel K; Warntges S; Bock J; Wagner C A; Sauter M; Waldegger S; Kandolf R; Lang F
CORPORATE SOURCE: Department of Molecular Pathology, Institute of Pathology, University of Tubingen, D-72076, Tubingen, Germany.
SOURCE: American journal of physiology. Gastrointestinal and liver physiology, (2000 Nov) 279 (5) G998-G1002.
Journal code: 100901227. ISSN: 0193-1857.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20020420
Entered Medline: 20001130

AB Transcript levels of the **human** serine/threonine **kinase** h-sgk have been found to be highest in pancreas. In the present study, localization and regulation of h-sgk transcription in pancreatic tissue were elucidated. As was apparent from radioactive in situ hybridization, most pancreatic acinar cells expressed high levels of h-sgk mRNA. h-sgk mRNA-positive cells were also found in ductal epithelia but not in pancreatic islets. In biopsy specimens from patients with pancreatitis, h-sgk mRNA levels were decreased in acinar cells but abundant in numerous mononuclear interstitial cells within areas of pancreatic necrosis and fibrosis. As shown by Northern blotting, h-sgk transcription in DAN-G pancreatic tumor cells is upregulated by osmotic cell shrinkage, serum, phorbol esters (phorbol 12,13-didecanoate), and Ca(2+) ionophore A-23187 and decreased by staurosporine and cAMP. In conclusion, h-sgk transcription is regulated not only by cell volume but also by serum, protein **kinase** C stimulation, cAMP, and increase of intracellular Ca(2+) activity. The **kinase** may participate not

only in normal function of exocrine pancreas but also in fibrosing pancreatitis.

L4 ANSWER 12 OF 19 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 2001067208 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11093030
TITLE: h-sgk serine-threonine protein **kinase** as transcriptional target of p38/MAP **kinase** pathway in HepG2 **human** hepatoma cells.
AUTHOR: Waldegger S; Gabrysch S; Barth P; Fillon S; Lang F
CORPORATE SOURCE: Institut fur Physiologie I, Gmelinstr. 5, D-72076 Tubingen, Germany.
SOURCE: Cellular physiology and biochemistry : international journal of experimental cellular physiology, biochemistry, and pharmacology, (2000) 10 (4) 203-8.
Journal code: 9113221. ISSN: 1015-8987.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20020420
Entered Medline: 20001222

AB The **human** serum and glucocorticoid dependent serine/threonine **kinase** h-sgk has previously been discovered as cell **volume regulated** gene. The present study has been performed to elucidate the involvement of p38-**kinase** in the transcriptional control of h-sgk by osmotic cell shrinkage. The p38-**kinase** has previously been cloned as the mammalian homologue of HOG1 **kinase**, which constitutes a part of the osmosensor in the yeast Saccharomyces cerevisiae. Phosphorylated (active) p38-**kinase** has been estimated with Western blotting, transcription of hsgk using Northern blotting. Both, increase of extracellular NaCl concentration by 50 mmol/l and addition of 10 micromol/l anisomycin increase phosphorylation of the p38-**kinase** within 5 to 10 minutes. h-sgk transcription is upregulated by addition of 50 mmol/l NaCl and by anisomycin (10 micromol/l), effects completely inhibited by the specific p38-**kinase** inhibitor, SB 203580 (10 micromol/l). In conclusion, the stimulation of h-sgk transcription by osmotic cell shrinkage is mediated by p38-**kinase**.

L4 ANSWER 13 OF 19 MEDLINE on STN
ACCESSION NUMBER: 2001067206 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11093028
TITLE: The shrinkage-activated Na(+) conductance of rat hepatocytes and its possible correlation to rENaC.
AUTHOR: Bohmer C; Wagner C A; Beck S; Moschen I; Melzig J; Werner A; Lin J T; Lang F; Wehner F
CORPORATE SOURCE: Max-Planck-Institut fur molekulare Physiologie, Abteilung Epithelphysiologie, Otto-Hahn-Str. 11, 44227 Dortmund, Germany.
SOURCE: Cellular physiology and biochemistry : international journal of experimental cellular physiology, biochemistry, and pharmacology, (2000) 10 (4) 187-94.
Journal code: 9113221. ISSN: 1015-8987.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20020420

Entered Medline: 20001222

AB At moderate cell shrinkage, activation of Na(+) channels is the most prominent mechanism of regulatory cell volume increase in rat hepatocytes. The amiloride sensitivity of these channels suggests a relation to the family of epithelial Na(+) channels (ENaCs). The present study was performed to determine the pharmacological profile of shrinkage-activated Na(+) channels and to test for ENaC expression in primary cultures of rat hepatocytes; in addition, the influence of the cell **volume regulated** serine/threonine **kinase** hSGK on activity and pharmacological profile of rENaC was examined in Xenopus oocytes. Conventional electrophysiology in hepatocytes reveals that the shrinkage-activated Na(+) channel is inhibited by amiloride and EIPA with IC(50) values of 6.0 and 0.12 micromol/l, respectively. Western blots and RT-PCR demonstrate that rat hepatocytes do express all three subunits (alpha, beta, gamma) of ENaC. Coexpression of hSGK with rENaC in Xenopus oocytes reveals that the **kinase** stimulates ENaC by a factor of 4. Moreover, hSGK decreases the affinity to amiloride (increase of IC(50) from 0.12 to 0.26 micromol/l) and increases the affinity to EIPA (decrease of IC(50) from 250 to 50 micromol/l). In conclusion, rat hepatocytes express ENaC, which is activated by the cell volume-sensitive **kinase** hSGK. ENaC may contribute to the Na(+) channels activated by osmotic cell shrinkage in hepatocytes, whereby the relatively low amiloride and high EIPA sensitivity of the channel could at least be partially due to modification by SGK, which decreases the amiloride and increases the EIPA sensitivity of ENaC.

L4 ANSWER 14 OF 19 MEDLINE on STN DUPLICATE 8

ACCESSION NUMBER: 1999458672 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10527936
TITLE: Osmotic cell swelling-induced ATP release mediates the activation of extracellular signal-regulated protein **kinase** (Erk)-1/2 but not the activation of osmo-sensitive anion channels.
AUTHOR: Van der Wijk T; De Jonge H R; Tilly B C
CORPORATE SOURCE: Department of Biochemistry, Cardiovascular Research Institute COEUR, Faculty of Medicine and Health Sciences, Erasmus University, P.O. Box 1738, 3000 DR Rotterdam, The Netherlands.
SOURCE: Biochemical journal, (1999 Nov 1) 343 Pt 3 579-86. Journal code: 2984726R. ISSN: 0264-6021.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000114
Last Updated on STN: 20000114
Entered Medline: 20000103

AB **Human** intestine 407 cells respond to hypo-osmotic stress by the rapid release of ATP into the extracellular medium. A difference in the time course of activation as well as in the sensitivity to cytochalasin B treatment and BAPTA-AM [1,2-bis-(2-aminophenoxy)ethane-N,N,N',N'-tetra-acetic acid acetoxymethyl ester] loading suggests that ATP leaves the cell through a pathway distinct from **volume-regulated** anion channels. To evaluate a putative role for nucleotides as autocrinic/paracrinic factors in osmotic signalling, the effects of extracellular ATP on the regulation of volume-sensitive anion channels as well as on the hypotonicity-induced activation of extracellular signal-regulated protein **kinases** (Erk-1/2) were investigated. Micromolar concentrations of ATP were unable to elicit an isotope efflux from (125)I(-)-loaded cells by itself, but strongly potentiated the hypotonicity-provoked anion efflux through a Ca(2+)-dependent mechanism. The order of potency of nucleotides (ATP = UTP = ATP[S] > ADP = AMP >>

adenosine = cAMP) indicated the involvement of P2Y(2) receptors. In contrast, millimolar concentrations of ATP markedly inhibited both the osmotically induced isotope efflux and whole-cell Cl(-) currents. Inhibition of whole-cell Cl(-) currents, not only by millimolar ATP but also by the purinoceptor antagonists suramin and reactive blue, was observed most prominently at depolarizing holding potentials, suggesting a direct interaction with volume-sensitive Cl(-) channels rather than interaction with purinoceptors. Both ATP and UTP, at submicromolar levels, were found to act as potent activators of Erk-1/2 in intestine 407 cells. Addition of the ATP hydrolase apyrase to the bath greatly reduced the hypotonicity-induced Erk-1/2 activation, but did not affect the swelling-induced isotope efflux or whole-cell Cl(-) currents. Furthermore, pre-treatment with suramin or reactive blue almost completely prevented the hypo-osmotic activation of Erk-1/2. The results indicate that extracellularly released ATP functions as an autocrinic/paracrinic factor that mediates hypotonicity-induced Erk-1/2 activation but does not serve as an activator of volume-sensitive compensatory Cl(-) currents.

L4 ANSWER 15 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:604791 HCAPLUS
DOCUMENT NUMBER: 129:213510
TITLE: The **human** homolog of the cell **volume regulated** protein **kinase** sgk and the gene encoding it
INVENTOR(S): Lang, Florian; Waldegger, Siegfried
PATENT ASSIGNEE(S): Dade Behring Marburg G.m.b.H., Germany
SOURCE: Eur. Pat. Appl., 15 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 861896	A2	19980902	EP 1998-101338	19980127
EP 861896	A3	19991020		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
DE 19708173	A1	19980903	DE 1997-19708173	19970228
CA 2224404	AA	19980828	CA 1998-2224404	19980226
US 6326181	B1	20011204	US 1998-31295	19980226
JP 10248566	A2	19980922	JP 1998-46565	19980227
US 2003003559	A1	20030102	US 2001-39	20011204

PRIORITY APPLN. INFO.: DE 1997-19708173 A 19970228
US 1998-31295 A3 19980226

AB The **human** gene for the cell **vol.-regulated kinase** sgk (serum and glucocorticoid-dependent **kinase**) is cloned and characterized. The enzyme can be used in the diagnosis and treatment of diseases associated with abnormal changes in cell vols. or macromol. crowding. Genes induced in HepG2 cells under hypertonic and hypotonic conditions were identified by RAP-PCR. A specific transcript that was expressed under hypertonic and hypotonic conditions was further characterized. The sequence of the full-length transcript had a 95% identity to a part of the rat sgk gene.

L4 ANSWER 16 OF 19 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN DUPLICATE 9
ACCESSION NUMBER: 1998305122 EMBASE
TITLE: Cloning of sgk serine-threonine protein **kinase** from shark rectal gland - A gene induced by hypertonicity and secretagogues.
AUTHOR: Waldegger S.; Barth P.; Forrest J.N. Jr.; Greger R.; Lang F.

CORPORATE SOURCE: S. Waldegger, Department of Physiology 1, University of Tubingen, Gmelinstr. 5, D-72076 Tubingen, Germany
SOURCE: Pflugers Archiv European Journal of Physiology, (1998) 436/4 (575-580).
Refs: 35
ISSN: 0031-6768 CODEN: PFLABK
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Recently, the cell-**volume-regulated** serine-threonine protein **kinase** h- sgk was cloned from a **human** hepatoma cell line. The sgk gene was shown to be induced by cell shrinkage in many different mammalian cell lines. In this study, two highly conserved serine-threonine protein **kinases**, sgk-1 and sgk- 2, were cloned from rectal gland tissue of the spiny dogfish (Squalus acanthias). Both **kinases** showed a distinct pattern of tissue specificity, with high expression levels in kidney, intestine, liver and heart. In rectal gland slices sgk-1 transcription was induced by exposure to hypertonic solution, reduction of the extracellular urea concentration, and addition of the secretagogues vasoactive intestinal polypeptide (VIP) and carbachol. The shark sgk-1 serine-threonine protein **kinase** may therefore provide a link between cell volume, Cl-secretion and protein phosphorylation state in shark rectal gland cells.

L4 ANSWER 17 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:547525 HCAPLUS
DOCUMENT NUMBER: 129:300487
TITLE: Electrophysiological properties of **volume-regulated** Cl- channels in intestinal epithelial cells
AUTHOR(S): Oiki, Shigetoshi; Kubo, Machiko; Okada, Yasunobu
CORPORATE SOURCE: Department of Cellular and Molecular Physiology, National Institute for Physiological Sciences, Okazaki, 444-8585, Japan
SOURCE: International Congress Series (1998), 1160(Cell Volume Regulation: The Molecular Mechanism and Volume Sensing Machinery), 125-129
CODEN: EXMDA4; ISSN: 0531-5131
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Regulation of their volume is a fundamental homeostatic function of animal cells. In a wide variety of cell types, the regulatory volume decrease which follows osmotic swelling is accomplished by parallel activation of K- and Cl- conductances followed by obligatory water efflux. Depletion of cytosolic ATP abolished swelling-induced activation of Cl- currents in **human** epithelial cells (Intestine 407 cells). The ATP role could be substituted by AMP-PNP, ATPγS, GTP and GTPγS. Blockers for protein **kinases** and protein phosphatases failed to affect swelling-activated Cl- currents. Thus, it is concluded that non-hydrolytic binding of ATP is involved in regulation of the volume-sensitive Cl- channel. The regulation by cytosolic ATP of the volume-sensitive Cl- channel may provide a fine tuning mechanism for cell volume regulation under osmotic or metabolic perturbation.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 18 OF 19 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 1998:81698 SCISEARCH
THE GENUINE ARTICLE: YR634
TITLE: Properties of **volume-regulated** anion

channels in mammalian cells
AUTHOR: Nilius B (Reprint); Eggermont J; Voets T; Buyse G; Manolopoulos V; Droogmans G
CORPORATE SOURCE: KATHOLIEKE UNIV LEUVEN, FYSIOL LAB, CAMPUS GASTHUISBERG, B-3000 LOUVAIN, BELGIUM (Reprint)
COUNTRY OF AUTHOR: BELGIUM
SOURCE: PROGRESS IN BIOPHYSICS & MOLECULAR BIOLOGY, (8 DEC 1997) Vol. 68, No. 1, pp. 69-119.
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD, ENGLAND OX5 1GB.
ISSN: 0079-6107.
DOCUMENT TYPE: General Review; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 328

L4 ANSWER 19 OF 19 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 95:115826 SCISEARCH
THE GENUINE ARTICLE: QE655
TITLE: VOLUME-ACTIVATED CHLORIDE CURRENTS ASSOCIATED WITH THE MULTIDRUG-RESISTANCE P-GLYCOPROTEIN
AUTHOR: HIGGINS C F (Reprint)
CORPORATE SOURCE: UNIV OXFORD, JOHN RADCLIFFE HOSP, NUFFIELD DEPT CLIN BIOCHEM, OXFORD OX3 9DU, ENGLAND (Reprint); UNIV OXFORD, JOHN RADCLIFFE HOSP, INST MOLEC MED, OXFORD OX3 9DU, ENGLAND
COUNTRY OF AUTHOR: ENGLAND
SOURCE: JOURNAL OF PHYSIOLOGY-LONDON, (JAN 1995) Vol. 482P, pp. S31-S36.
ISSN: 0022-3751.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 40
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The ability to regulate volume is an important property of most, if not all cells. In epithelial cells, amongst others, cell volume-activated chloride channels are central to this response. The molecular identities of these channels are not yet known. Expression of the **human** multidrug resistance P-glycoprotein (P-gp) has been associated with cell **volume-regulated** chloride currents, although the nature of this association is the subject of debate. Recent data indicate that P-gp acts by regulating the activation of an endogenous channel protein. In this review, evidence associating P-gp with cell volume-activated chloride currents, and the possible mechanisms by which this might be achieved, are discussed.

=> d his

(FILE 'HOME' ENTERED AT 09:30:06 ON 28 MAY 2004)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS' ENTERED AT 09:30:37 ON 28 MAY 2004
L1 881 S VOLUME(A)REGULATED
L2 157 S L1 AND KINASE?
L3 49 S HUMAN AND L2
L4 19 DUP REM L3 (30 DUPLICATES REMOVED)

=> s "h-sgk"
L5 39 "H-SGK"

=> s dup rem l5
MISSING OPERATOR REM L5

The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6 20 DUP REM L5 (19 DUPLICATES REMOVED)

=> s l6 and kinase?
L7 20 L6 AND KINASE?

=> d 1-20 ibib ab

L7 ANSWER 1 OF 20 MEDLINE on STN
ACCESSION NUMBER: 2001067208 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11093030
TITLE: **h-sgk** serine-threonine protein **kinase** as transcriptional target of p38/MAP **kinase** pathway in HepG2 human hepatoma cells.
AUTHOR: Waldegger S; Gabrysch S; Barth P; Fillon S; Lang F
CORPORATE SOURCE: Institut fur Physiologie I, Gmelinstr. 5, D-72076 Tubingen, Germany.
SOURCE: Cellular physiology and biochemistry : international journal of experimental cellular physiology, biochemistry, and pharmacology, (2000) 10 (4) 203-8.
Journal code: 9113221. ISSN: 1015-8987.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20020420
Entered Medline: 20001222

AB The human serum and glucocorticoid dependent serine/threonine **kinase h-sgk** has previously been discovered as cell volume regulated gene. The present study has been performed to elucidate the involvement of p38-**kinase** in the transcriptional control of **h-sgk** by osmotic cell shrinkage. The p38-**kinase** has previously been cloned as the mammalian homologue of HOG1 **kinase**, which constitutes a part of the osmosensor in the yeast Saccharomyces cerevisiae. Phosphorylated (active) p38-**kinase** has been estimated with Western blotting, transcription of hsgk using Northern blotting. Both, increase of extracellular NaCl concentration by 50 mmol/l and addition of 10 micromol/l anisomycin increase phosphorylation of the p38-**kinase** within 5 to 10 minutes. **h-sgk** transcription is upregulated by addition of 50 mmol/l NaCl and by anisomycin (10 micromol/l), effects completely inhibited by the specific p38-**kinase** inhibitor, SB 203580 (10 micromol/l). In conclusion, the stimulation of **h-sgk** transcription by osmotic cell shrinkage is mediated by p38-**kinase**.

L7 ANSWER 2 OF 20 MEDLINE on STN
ACCESSION NUMBER: 2001034894 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11052997
TITLE: Expression of cell volume-regulated **kinase h-sgk** in pancreatic tissue.
AUTHOR: Klingel K; Warntges S; Bock J; Wagner C A; Sauter M; Waldegger S; Kandolf R; Lang F
CORPORATE SOURCE: Department of Molecular Pathology, Institute of Pathology, University of Tubingen, D-72076, Tubingen, Germany.
SOURCE: American journal of physiology. Gastrointestinal and liver physiology, (2000 Nov) 279 (5) G998-G1002.

Journal code: 100901227. ISSN: 0193-1857.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20020420
Entered Medline: 20001130

AB Transcript levels of the human serine/threonine **kinase h -sgk** have been found to be highest in pancreas. In the present study, localization and regulation of **h-sgk** transcription in pancreatic tissue were elucidated. As was apparent from radioactive in situ hybridization, most pancreatic acinar cells expressed high levels of **h-sgk** mRNA. **h-sgk** mRNA-positive cells were also found in ductal epithelia but not in pancreatic islets. In biopsy specimens from patients with pancreatitis, **h-sgk** mRNA levels were decreased in acinar cells but abundant in numerous mononuclear interstitial cells within areas of pancreatic necrosis and fibrosis. As shown by Northern blotting, **h-sgk** transcription in DAN-G pancreatic tumor cells is upregulated by osmotic cell shrinkage, serum, phorbol esters (phorbol 12,13-didecanoate), and Ca(2+) ionophore A-23187 and decreased by staurosporine and cAMP. In conclusion, **h-sgk** transcription is regulated not only by cell volume but also by serum, protein **kinase** C stimulation, cAMP, and increase of intracellular Ca(2+) activity. The **kinase** may participate not only in normal function of exocrine pancreas but also in fibrosing pancreatitis.

L7 ANSWER 3 OF 20 MEDLINE on STN
ACCESSION NUMBER: 1999238882 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10220500
TITLE: **h-sgk** serine-threonine protein **kinase** gene as transcriptional target of transforming growth factor beta in human intestine.
AUTHOR: Waldegger S; Klingel K; Barth P; Sauter M; Rfer M L; Kandolf R; Lang F
CORPORATE SOURCE: Institute of Physiology, University of Tubingen, Tubingen, Germany.. florian.lang@uni-tuebingen.de
SOURCE: Gastroenterology, (1999 May) 116 (5) 1081-8.
Journal code: 0374630. ISSN: 0016-5085.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990618
Last Updated on STN: 20020420
Entered Medline: 19990607

AB BACKGROUND & AIMS: Recently, the immediate early gene **h-sgk** was cloned as a hypertonicity-induced gene from human hepatoma cells. The aim of this study was to localize **h-sgk** messenger RNA (mRNA) expression in normal and inflamed intestinal mucosa and to identify potential transcriptional regulators. METHODS: **h -sgk** mRNA in small intestinal mucosa from healthy persons and patients with Crohn's disease was determined by in situ hybridization. Transcriptional regulation was studied by Northern blot analysis of total RNA isolated from cultured human Intestine 407, U937, and HepG2 cells. RESULTS: In normal ileum, **h-sgk** mRNA was selectively localized to the apical villus enterocytes, whereas no staining was detected in crypt cells. In Crohn's disease, enterocytes of the crypts expressed **h-sgk** and abundant **h-sgk** positive inflammatory cells appeared in the lamina propria. Combined

h-sgk in situ hybridization and immunohistochemical analysis of CD68 antigen expression identified a part of these cells as macrophages. In addition to spatial correlation of transforming growth factor (TGF)-beta1 protein and **h-sgk** mRNA expression, **h-sgk** transcription in human Intestine 407 and HepG2 cells as well as in U937 monocytes/macrophages was strongly induced by TGF-beta1 in vitro. CONCLUSIONS: **h-sgk** expression in normal and inflamed intestinal mucosa may be regulated by TGF-beta1 and may contribute to the pleiotropic actions of TGF-beta1 in mucosal cell populations.

L7 ANSWER 4 OF 20 MEDLINE on STN
ACCESSION NUMBER: 97272242 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9114008
TITLE: Cloning and characterization of a putative human serine/threonine protein **kinase** transcriptionally modified during anisotonic and isotonic alterations of cell volume.
AUTHOR: Waldegger S; Barth P; Raber G; Lang F
CORPORATE SOURCE: Physiologisches Institut I der Eberhard-Karls-Universitat, D-72076 Tubingen, Germany.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1997 Apr 29) 94 (9) 4440-5. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-Y10032
ENTRY MONTH: 199705
ENTRY DATE: Entered STN: 19970609
Last Updated on STN: 20020420
Entered Medline: 19970527

AB Hepatic metabolism and gene expression are among other regulatory mechanisms controlled by the cellular hydration state, which changes rapidly in response to anisotonicity, concentrative substrate uptake, oxidative stress, and under the influence of hormones such as insulin and glucagon. Differential screening for cell volume sensitive transcripts in a human hepatoma cell line revealed a gene for a putative serine/threonine **kinase**, **h-sgk**, which has 98% sequence identity to a serum- and glucocorticoid regulated **kinase**, sgk, cloned from a rat mammary tumor cell line. **h-sgk** transcript levels were strongly altered during anisotonic and isotonic cell volume changes. Within 30 min **h-sgk** RNA was, independent of de novo protein synthesis, induced upon cell shrinkage and, due to a complete stop in **h-sgk** transcription, reduced upon cell swelling. Comparable changes of sgk transcript levels were observed in a renal epithelial cell line. **h-sgk** mRNA was detected in all human tissues tested, with the highest levels in pancreas, liver, and heart. The putative serine/threonine protein **kinase** **h-sgk** may provide a functional link between the cellular hydration state and metabolic control.

L7 ANSWER 5 OF 20 EMBASE on STN
ACCESSION NUMBER: 2001126681 EMBASE
TITLE: Expression and localization of serum/glucocorticoid-induced **kinase** in the rat ovary: Relation to follicular growth and differentiation.
AUTHOR: Alliston T.N.; Gonzalez-Robayna I.J.; Buse P.; Firestone G.L.; Richards J.S.
CORPORATE SOURCE: Dr. J.S. Richards, Department of Molecular Biology, Baylor College of Medicine, Houston, TX 77030, United States. joanner@bcm.tmc.edu

SOURCE: Endocrinology, (2000) 141/1 (385-395).
Refs: 54
ISSN: 0013-7227 CODEN: ENDOAO
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
010 Obstetrics and Gynecology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Expression of serum/glucocorticoid-inducible **kinase** (Sgk), one member of an inducible serine/threonine **kinase** family, is induced by FSH/cAMP in rat granulosa cells cultured in defined medium. The FSH-stimulated pattern of sgk expression is biphasic, and transcriptional activation of the sgk gene depends on an intact Sp1/Sp3 binding site within the proximal promoter. To determine whether sgk was expressed in a hormone-dependent and physiologically relevant manner in vivo, the cellular levels of sgk messenger RNA (mRNA) and protein as well as the subcellular localization of this **kinase** were analyzed in ovaries containing follicles and corpora lutea at specific stages of differentiation. To stimulate follicular development and luteinization, hypophysectomized (H) rats were treated with estradiol (E; HE) and FSH (FSH; HEF) followed by hCG (hCG; HEF/hCG). To analyze Sgk in functional corpora lutea, PRL was administered to HEF/hCG rats, or ovaries of pregnant rats were obtained on day 7, 15, or 22 of gestation. In situ hybridization indicated that sgk mRNA was low/undetectable in granulosa cells of H and HE rats. An acute in jection (iv) of FSH to HE rats rapidly increased sgk mRNA at 2 and 8 **h**. **Sgk** mRNA was also elevated in granulosa cells of preovulatory follicles of HEF rats and in luteal cells of HEF/hCG and pregnant rats. Northern blots and Western blots confirmed the in situ hybridization data, indicating that the amount and cellular localization Sgk protein were related to that of sgk mRNA. When the subcellular localization of this **kinase** was analyzed by immunohistochemistry, Sgk protein was nuclear in granulosa cells and some thecal cells of large preovulatory follicles. In contrast, Sgk protein was cytoplasmic in luteal cells as well as some cells within the stromal compartment. Intense immunostaining was also observed in oocytes present in primordial follicles, but not in growing follicles. Collectively, these results show that FSH and LH stimulate marked increases in the cellular content of Sgk, as well as dramatic changes in the subcellular distribution of this **kinase**. The specific nuclear vs. cytoplasmic compartmentalization of Sgk in granulosa cells and luteal cells, respectively, indicates that Sgk controls distinct functions in proliferative vs. terminally differentiated granulosa cells.

L7 ANSWER 6 OF 20 EMBASE
on STN
ACCESSION NUMBER: 1998305122 EMBASE
TITLE: Cloning of sgk serine-threonine protein **kinase** from shark rectal gland - A gene induced by hypertonicity and secretagogues.
AUTHOR: Waldegger S.; Barth P.; Forrest J.N. Jr.; Greger R.; Lang F.
CORPORATE SOURCE: S. Waldegger, Department of Physiology 1, University of Tubingen, Gmelinstr. 5, D-72076 Tubingen, Germany
SOURCE: Pflugers Archiv European Journal of Physiology, (1998) 436/4 (575-580).
Refs: 35
ISSN: 0031-6768 CODEN: PFLABK
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
029 Clinical Biochemistry
LANGUAGE: English

SUMMARY LANGUAGE: English

AB Recently, the cell-volume-regulated serine-threonine protein **kinase h- sgk** was cloned from a human hepatoma cell line. The sgk gene was shown to be induced by cell shrinkage in many different mammalian cell lines. In this study, two highly conserved serine-threonine protein **kinases**, sgk-1 and sgk- 2, were cloned from rectal gland tissue of the spiny dogfish (Squalus acanthias). Both **kinases** showed a distinct pattern of tissue specificity, with high expression levels in kidney, intestine, liver and heart. In rectal gland slices sgk-1 transcription was induced by exposure to hypertonic solution, reduction of the extracellular urea concentration, and addition of the secretagogues vasoactive intestinal polypeptide (VIP) and carbachol. The shark sgk-1 serine-threonine protein **kinase** may therefore provide a link between cell volume, Cl-secretion and protein phosphorylation state in shark rectal gland cells.

L7 ANSWER 7 OF 20 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:74113 BIOSIS
DOCUMENT NUMBER: PREV200200074113
TITLE: Cell volume-regulated human **kinase h-sgk**.
AUTHOR(S): Lang, Florian [Inventor, Reprint author]; Waldegger, Siegfried [Inventor]
CORPORATE SOURCE: Im Rotbad 52, 72076 Tubingen, Germany
PATENT INFORMATION: US 6326181 December 04, 2001
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Dec. 4, 2001) Vol. 1253, No. 1. ftp://ftp.uspto.gov/pub/patdata/. e-file. CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jan 2002
Last Updated on STN: 25 Feb 2002

AB The present invention relates to the cloning and characterization of a human serine/threonine **kinase** (**h-sgk**: serum and glucocorticoid dependent **kinase**). The invention furthermore relates to reagents for diagnosing conditions associated with a change in cell volume and/or in "macromolecular crowding" in the body, such as, for example, hypernatremia, hyponatremia, diabetes mellitus, renal failure, hypercatabolism, hepatic encephalopathy, inflammation and microbial or viral infections. The present invention additionally relates to pharmaceuticals comprising the **h-sgk**, nucleic acids which code for the **h-sgk**, or receptors, in particular antibodies, which specifically bind to the **h-sgk**.

L7 ANSWER 8 OF 20 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:358201 BIOSIS
DOCUMENT NUMBER: PREV200100358201
TITLE: Association between inflammation and expression of human serine threonine **kinase** (**h-sgk**) in fetal and neonatal lung tissue.
AUTHOR(S): Speer, Christian P. [Reprint author]; Schmidt, Beate [Reprint author]; Cao, Lei [Reprint author]; Klingel, Karin; Mackensen-Haen, Susanne; Lang, Florian
CORPORATE SOURCE: University Children's Hospital, Wuerzburg, Germany
SOURCE: Biology of the Neonate, (May, 2001) Vol. 80, No. Supplement 1, pp. 35. print.
Meeting Info.: Proceedings of the 16th International Workshop on Surfactant Replacement. Edinburgh, Scotland. June 02-04, 2001.
CODEN: BNEOBV. ISSN: 0006-3126.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English

ENTRY DATE: Entered STN: 2 Aug 2001
Last Updated on STN: 19 Feb 2002

L7 ANSWER 9 OF 20 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:244741 BIOSIS
DOCUMENT NUMBER: PREV200100244741
TITLE: All three isoforms of human serum and glucocorticoid dependent **kinase** (**h-SGK**) upregulate voltage-gated potassium channels endogenously expressed in HEK293 cells.
AUTHOR(S): Fillon, S. [Reprint author]; Gamper, N. [Reprint author]; Huber, S. M. [Reprint author]; Feng, Y. X. [Reprint author]; Friedrich, B. [Reprint author]; Kobayashi, T.; Cohen, P.; Lang, F. [Reprint author]
CORPORATE SOURCE: Institute of Physiology, University of Tuebingen, Tuebingen, Germany
SOURCE: Pfluegers Archiv European Journal of Physiology, (2001) Vol. 441, No. 6 Supplement, pp. R182. print.
Meeting Info.: Joint Congress of the Scandinavian and the German Physiological Societies. Berlin, Germany. March 10-13, 2001.
CODEN: PFLABK. ISSN: 0031-6768.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 May 2001
Last Updated on STN: 19 Feb 2002

L7 ANSWER 10 OF 20 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:527010 BIOSIS
DOCUMENT NUMBER: PREV199900527010
TITLE: Cell volume regulatory **kinase h-sgk** in the pathophysiology of diabetic nephropathy.
AUTHOR(S): Lang, Florian [Reprint author]; Wagner, Carsten A. [Reprint author]; Broer, Stefan [Reprint author]; Melzig, Joerg [Reprint author]; Waldegger, Siegfried [Reprint author]; Steuer, Silvia; Lanzendorfer, Martina; Klingel, Karin; Kandolf, Reinhard; Heidland, August; Capasso, Giovambattista; Massry, Shaul G.; Risler, Teut
CORPORATE SOURCE: Department of Physiology, University of Tuebingen, Tuebingen, Germany
SOURCE: Journal of the American Society of Nephrology, (Sept., 1999) Vol. 10, No. PROGRAM AND ABSTR. ISSUE, pp. 685A. print.
Meeting Info.: 32nd Annual Meeting of the American Society of Nephrology. Miami Beach, Florida, USA. November 1-8, 1999. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1999
Last Updated on STN: 10 Dec 1999

L7 ANSWER 11 OF 20 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
ACCESSION NUMBER: 1998-10366 BIOTECHDS
TITLE: New nucleic acid encoding cell-volume regulating **kinase h-sgk** and related proteins
;
enzyme and protein used for diagnosis and therapy of condition related to cell-volume change
AUTHOR: Lang F; Waldegger S

PATENT ASSIGNEE: Dade-Behring-Marburg
LOCATION: Marburg, Germany.
PATENT INFO: EP 861896 2 Sep 1998
APPLICATION INFO: EP 1998-101338 27 Jan 1998
PRIORITY INFO: DE 1997-1008173 28 Feb 1997
DOCUMENT TYPE: Patent
LANGUAGE: German
OTHER SOURCE: WPI: 1998-449109 [39]

AB A nucleic acid (A) that encodes the human cell-volume regulating serum and glucocorticoid-dependent **kinase** (**h-sgk**) with a given 431 amino acid protein sequence is claimed. (A) has a given 2,370 bp nucleotide sequence. Also claimed are nucleic acids that hybridize with (A) under stringent conditions and encode an active cell-volume regulating **kinase**, the transcription of which is not induced by fetal cattle-serum or glucocorticoids. Alternatively it can encode a **kinase** that is not identical with rat-sgk. The claims also cover polynucleotide fragments consisting of bases 980-1,480 of the given sequence that encodes an immunogenic fragment of **h-sgk**. The claims extend to recombinant **h-sgk**, and receptors that specifically bind to **h-sgk**. The new nucleic acids are used to detect (A) by Northern blotting and hybridization. The protein **h-sgk** can be used to detect receptors which can be used to detect and quantify **h-sgk** in immunoassays. This has application in diagnosis and therapy of conditions associated with cell-volume changes, including hyper-and hypo-natriemia, diabetes mellitus, fructose intolerance, Alzheimer disease, etc. (15pp)

L7 ANSWER 12 OF 20 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2004:390716 SCISEARCH
THE GENUINE ARTICLE: 814KP
TITLE: Association of the serum and glucocorticoid regulated **kinase** (sgk1) gene with QT interval
AUTHOR: Busjahn A; Seebohm G; Maier G; Toliat M R; Nurnberg P; Aydin A; Luft F C (Reprint); Lang F
CORPORATE SOURCE: HELIOS Kliniken Berlin, Franz Volhard Clin, Wiltberg Str 50, D-13125 Berlin, Germany (Reprint); HELIOS Kliniken Berlin, Franz Volhard Clin, D-13125 Berlin, Germany; Humboldt Univ, Fac Med Charite, Max Delbruck Ctr Mol Med, Berlin, Germany; Univ Tubingen, Dept Physiol, D-72074 Tubingen, Germany; Max Delbruck Ctr Mol Med, Gene Mapping Ctr, Berlin, Germany
COUNTRY OF AUTHOR: Germany
SOURCE: CELLULAR PHYSIOLOGY AND BIOCHEMISTRY, (APR 2004) Vol. 14, No. 3, pp. 135-142.
Publisher: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.
ISSN: 1015-8987.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 65

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The serum and glucocorticoid inducible **kinase** (SGK1) is well known to up-regulate the renal epithelial Na+ channel ENaC. Excessive SGK1 activity would be expected to cause renal Na+ retention and blood pressure increase. Certain polymorphisms of the SGK1 gene (E8CC/CT; 16CC) are indeed associated with moderately enhanced blood pressure. We have recently disclosed another function of SGK1, i.e. the stimulation of the slowly activating K+ channel KCNE1/KCNQ1. Among the functions of this channel is the repolarisation of cardiac myocytes. Accordingly, defective KCNE1 and/or KCNQ1 lead to long QT syndrome, a disorder causing fainting and sudden cardiac death. In the present study we demonstrate that coexpression of SGK1 in Xenopus oocytes increases KCNQ1/KCNE1 induced current without significantly altering voltage dependence, activation and

deactivation kinetics. To test for the relevance of SGK1 in human cardiac repolarization, we analysed the ECG of monozygotic (MZ) (126 pairs) and dizygotic (DZ) (70 pairs) twin subjects and parents of DZ twins. The E8CC/CT;16CC polymorphism was indeed significantly ($p < 0.025$) associated with shortened age and gender corrected QT interval. No significant differences were observed in any other ECG parameter, including heart rate, P, PQ and QRS. We conclude that the regulation of KCNE1/KCNQ1 by SGK1 is similarly relevant for the repolarization of cardiac myocytes as for regulation of renal ENaC activity and blood pressure control. Copyright c 2004 S. Karger AG, Basel.

L7 ANSWER 13 OF 20 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2003:401550 SCISEARCH
THE GENUINE ARTICLE: 674VM
TITLE: Stimulus-dependent regulation of serum and glucocorticoid inducible protein **kinase** (SGK) transcription, subcellular localization and enzymatic activity
AUTHOR: Firestone G L (Reprint); Giampaolo J R; O'Keeffe B A
CORPORATE SOURCE: Univ Calif Berkeley, Dept Mol & Cell Biol, 591 LSA, Berkeley, CA 94720 USA (Reprint); Univ Calif Berkeley, Dept Mol & Cell Biol, Berkeley, CA 94720 USA; Univ Calif Berkeley, Canc Res Lab, Berkeley, CA 94720 USA
COUNTRY OF AUTHOR: USA
SOURCE: CELLULAR PHYSIOLOGY AND BIOCHEMISTRY, (APR 2003) Vol. 13, No. 1, pp. 1-12.
Publisher: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.
ISSN: 1015-8987.
DOCUMENT TYPE: General Review; Journal
LANGUAGE: English
REFERENCE COUNT: 79

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We originally discovered the serum and glucocorticoid inducible protein **kinase**, SGK, as a novel protein **kinase** that is under acute transcriptional control by serum and glucocorticoids. An expanding set of cell surface receptor, nuclear receptor, and cellular stress pathways has been shown to target SGK, which has implicated this regulated signaling molecule in a variety of biological functions. Compared to most other protein **kinases**, a distinguishing feature of SGK is the stringent stimulus-dependent regulation of its transcription, subcellular localization and enzymatic activity. In addition, SGK expression is regulated during discrete developmental stages, and during normal and abnormal physiological function. An analysis of the SGK promoter reveals many potential transcription factor sites that potentially account for the stimulus-dependent changes in SGK transcript expression observed in a variety of cell systems, although, the direct stimulus regulation of SGK promoter activity has been established only for glucocorticoids, p53 tumor suppressor protein, hyperosmotic stress and follicle stimulating hormone. In the systems tested to date, hormones, growth factors and environmental cues induce expression of a catalytically active SGK. It is now well established that the enzymatic activity of SGK is controlled by the PI 3-**kinase** cascade which produces a hyperphosphorylated active SGK. A critical third level of regulation is the stimulus-dependent control of SGK subcellular localization. The nuclear-cytoplasmic shuttling of SGK is regulated by a nuclear localization signal (NLS) that binds to the importin-alpha nuclear import receptor. Modeling of the 3-D structure of the central region of SGK that includes the **kinase** domain predicts that the MLS is located at an external surface of the molecule. Thus, multiple signal transduction pathways converge on SGK to control its availability, function and access to its substrates and nonsubstrate targets. Copyright (C) 2003 S. Karger AG, Basel.

L7 ANSWER 14 OF 20 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2003:241577 SCISEARCH

THE GENUINE ARTICLE: 654HJ
TITLE: Activation of Na+/K+-ATPase by the serum and glucocorticoid-dependent **kinase** isoforms
AUTHOR: Henke G; Setiawan I; Bohmer C; Lang F (Reprint)
CORPORATE SOURCE: Univ Tubingen, Inst Physiol, Gmelinstr 5, D-72076 Tubingen, Germany (Reprint); Univ Tubingen, Inst Physiol, D-72076 Tubingen, Germany
COUNTRY OF AUTHOR: Germany
SOURCE: KIDNEY & BLOOD PRESSURE RESEARCH, (DEC 2002) Vol. 25, No. 6, pp. 370-374.
Publisher: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.
ISSN: 1420-4096.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 49
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Background/Aim: Expression of the constitutively active form of serum and glucocorticoid-dependent **kinase** ((S422D)SGK1) in Xenopus oocytes has recently been shown to upregulate endogenous Na+/K+-ATPase activity, an effect presumably participating in the regulation of cellular K+ uptake and transepithelial Na+ transport. SGK1 and the two isoforms SGK2 and SGK3 are stimulated by insulin and insulin-like growth factor-1 (IGF-1), which have been shown to enhance Na+/K+-ATPase activity in a variety of cells. The present experiments have been performed to elucidate whether or not wild-type SGK1, SGK2 and SGK3 are similar to (S422D)SGK1 in being effective regulators of Na+/K+-ATPase. Methods: To this end, dual-electrode voltage clamp experiments were performed in Xenopus oocytes injected either with water or with mRNA of constitutively active (S422D)SGK1 and wild-type SGK1, SGK2 or SGK3. Na+/K+-ATPase activity was estimated from the outward-directed current created by readdition of extracellular K+ in the presence of K+ channel blocker Ba2+ following a 10-min exposure to K+-free extracellular fluid. Results: The outward-directed current was fully abolished by incubation with 1 mM ouabain and was significantly larger in oocytes expressing (S422D)SGK1, SGK1, SGK2 or SGK3, as compared to those injected with water. Conclusion: The stimulating effect of SGK1 on the Xenopus oocyte Na+/K+-ATPase is mimicked by the isoforms SGK2 and SGK3. Thus, all three **kinases** may participate in the regulation of Na+/K+-ATPase activity by hormones such as insulin and IGF-1. Copyright (C) 2002 S. Karger AG, Basel.

L7 ANSWER 15 OF 20 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2003:198707 SCISEARCH
THE GENUINE ARTICLE: 648BF
TITLE: Expression of the serum- and glueocorticoid-inducible protein **kinase**, Sgk, is a cell survival response to multiple types of environmental stress stimuli in mammary epithelial cells
AUTHOR: Leong M L L; Maiyar A C; Kim B; O'Keeffe B A; Firestone G L (Reprint)
CORPORATE SOURCE: Univ Calif Berkeley, Dept Mol & Cell Biol, 591 LSA, Berkeley, CA 94720 USA (Reprint); Univ Calif Berkeley, Dept Mol & Cell Biol, Berkeley, CA 94720 USA; Univ Calif Berkeley, Canc Res Lab, Berkeley, CA 94720 USA
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (21 FEB 2003) Vol. 278, No. 8, pp. 5871-5882.
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3996 USA.
ISSN: 0021-9258.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 86
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The effects of multiple stress stimuli on the cellular utilization of the serum- and glucocorticoid-inducible protein **kinase** (Sgk) were examined in NMuMg mammary epithelial cells exposed to hyperosmotic stress induced by the organic osmolyte sorbitol, heat shock, ultraviolet irradiation, oxidative stress induced by hydrogen peroxide, or to dexamethasone, a synthetic glucocorticoid that represents a general class of physiological stress hormones. Each of the stress stimuli induced Sgk protein expression with differences in the kinetics and duration of induction and in subcellular localization. The environmental stresses, but not dexamethasone, stimulated Sgk expression through a p38/ MAPK-dependent pathway. In each case, a hyperphosphorylated active Sgk protein was produced under conditions in which Akt, the close homolog of Sgk, remained in its non-phosphorylated state. Ectopic expression of wild type Sgk or of the T256D/S422D mutant Sgk that mimics phosphorylation conferred protection against stress-induced cell death in NMuMg cells. In contrast, expression of the T256A/S422A Sgk phosphorylation site mutant has no effect on cell survival. Sgk is known to phosphorylate and negatively regulate proapoptotic forkhead transcription factor FKHRL1. The environmental stress stimuli that induce Sgk, but not dexamethasone, strongly inhibited the nuclear transcriptional activity and increased the cytoplasmic retention of FKHRL1. Also, the conditional IPTG inducible expression of wild type Sgk, but not of the **kinase** dead T256A mutant Sgk, protected Con8 mammary epithelial tumor cells from serum starvation-induced apoptosis. Taken together, our study establishes that induction of enzymatically active Sgk functions as a key cell survival component in response to different environmental stress stimuli.

L7 ANSWER 16 OF 20 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2002:289276 SCISEARCH
THE GENUINE ARTICLE: 536XJ
TITLE: Expression of the serine/threonine **kinase** hSGK1 in chronic viral hepatitis
AUTHOR: Fillon S; Klingel K; Warntges S; Sauter M; Gabrysch S; Pestel S; Tanneur V; Waldegger S; Zipfel A; Viebahn R; Broer S; Kandolf R; Lang F (Reprint)
CORPORATE SOURCE: Univ Tubingen, Inst Physiol, Dept Physiol, Gmelinstr 5, D-72076 Tubingen, Germany (Reprint); Univ Tubingen, Inst Physiol, Dept Physiol, D-72076 Tubingen, Germany; Univ Tubingen, Dept Mol Pathol, D-72076 Tubingen, Germany; Univ Dusseldorf, Dept Internal Med, D-4000 Dusseldorf, Germany; Univ Tubingen, Dept Surg, D-72076 Tubingen, Germany; Australian Natl Univ, Sch Biochem & Mol Biol, Canberra, ACT, Australia
COUNTRY OF AUTHOR: Germany; Australia
SOURCE: CELLULAR PHYSIOLOGY AND BIOCHEMISTRY, (DEC 2002) Vol. 12, No. 1, pp. 47-54.
Publisher: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.
ISSN: 1015-8987.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 37
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The human serine/threonine **kinase** hSGK1 is expressed ubiquitously with highest transcript levels in pancreas and liver. This study has been performed to determine the hSGK1 distribution in normal liver and its putative role in fibrosing liver disease. HSGK1-localization was determined by in situ hybridization, regulation of hSGK1-transcription by Northern blotting, fibronectin synthesis and hSGK1 phosphorylation by Western blotting. In normal liver hSGK1 was mainly transcribed by Kupffer cells. In liver tissue from patients with chronic viral hepatitis, hSGK1 transcript levels were excessively high in numerous activated Kupffer cells and inflammatory cells localized within fibrous septum formations. HSGK1 transcripts were also detected in activated hepatic stellate cells.

Accordingly, Western blotting revealed that tissue from fibrotic liver expresses excessive hSGK1 protein as compared to normal liver. TGF-beta1 (2 ng/ml) increases hSGK1 transcription in both human U937 macrophages and HepG2 hepatoma cells. H2O2 (0.3 mM) activated hSGK1 and increased fibronectin formation in HepG2 cells overexpressing hSGK1 but not in HepG2 cells expressing the inactive mutant hSGK1(K127R). In conclusion hSGK1 is upregulated by TGF-beta1 during hepatitis and may contribute to enhanced matrix formation during fibrosing liver disease. Copyright (C) 2002 S. Karger AG, Basel.

L7 ANSWER 17 OF 20 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2002:246823 SCISEARCH
THE GENUINE ARTICLE: 530ND
TITLE: Cerebral localization and regulation of the cell volume-sensitive serum- and glucocorticoid-dependent **kinase** SGK1
AUTHOR: Warntges S; Friedrich B; Henke G; Duranton C; Lang P A; Waldegger S; Meyermann R; Kuhl D; Speckmann E J; Obermuller N; Witzgall R; Mack A F; Wagner H J; Wagner C A; Broer S; Lang F (Reprint)
CORPORATE SOURCE: Univ Tubingen, Inst Physiol, Gmelinstr 5, D-72076 Tubingen, Germany (Reprint); Univ Tubingen, Inst Physiol, D-72076 Tubingen, Germany; Univ Tubingen, Dept Brain Res, D-72076 Tubingen, Germany; Univ Hamburg, Zentrum Mol Neurobiol, Hamburg, Germany; Univ Munster, Dept Physiol, D-4400 Munster, Germany; Univ Heidelberg, Dept Anat, D-6900 Heidelberg, Germany; Univ Tubingen, Dept Anat, D-72076 Tubingen, Germany; Yale Univ, Dept Cellular & Mol Physiol, New Haven, CT USA
COUNTRY OF AUTHOR: Germany; USA
SOURCE: PFLUGERS ARCHIV-EUROPEAN JOURNAL OF PHYSIOLOGY, (FEB 2002) Vol. 443, No. 4, pp. 617-624.
Publisher: SPRINGER-VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010 USA.
ISSN: 0031-6768.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 42
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The serum- and glucocorticoid-dependent **kinase** SGK1 is regulated by alterations of cell volume, whereby cell shrinkage increases and cell swelling decreases the transcription, expression and activity of SGK1. The **kinase** is expressed in all human tissues studied including the brain. The present study was performed to localize the sites of SGK1 transcription in the brain, to elucidate the influence of the hydration status on SGK1 transcription and to explore the functional significance of altered SGK1 expression. Northern blot analysis of human brain showed SGK1 to be expressed in all cerebral structures examined: amygdala, caudate nucleus, corpus callosum, hippocampus, substantia nigra, subthalamic nucleus and thalamus. In situ hybridization and immunohistochemistry in the rat revealed increased expression of SGK1 in neurons of the hippocampal area CA3 after dehydration, compared with similar slices from brains of euvolaemic rats. Additionally, several oligodendrocytes, a few microglial cells, but no astrocytes, were positive for SGK1. The abundance of SGK1 mRNA in the temporal lobe, including hippocampus, was increased by dehydration and SGK1 transcription in neuroblastoma cells was stimulated by an increase of extracellular osmolarity. Co-expression studies in Xenopus laevis oocytes revealed that SGK1 markedly increased the activity of the neuronal K+ channel Kv1.3. As activation of K+ channels modifies excitation of neuronal cells, SGK1 may participate in the regulation of neuronal excitability.

L7 ANSWER 18 OF 20 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2001:922107 SCISEARCH

THE GENUINE ARTICLE: 490MJ
TITLE: Cell volume regulatory mechanisms in progression of renal disease
AUTHOR: Warntges S; Grone H J; Capasso G; Lang F (Reprint)
CORPORATE SOURCE: Univ Tubingen, Inst Physiol, Gmelinstr 5, D-76072 Tubingen, Germany (Reprint); Univ Tubingen, Dept Physiol, Tubingen, Germany
COUNTRY OF AUTHOR: Germany
SOURCE: JOURNAL OF NEPHROLOGY, (SEP-OCT 2001) Vol. 14, No. 5, pp. 319-326.
Publisher: WICHTIG EDITORE, 72/74 VIA FRIULI, 20135 MILAN, ITALY.
ISSN: 1121-8428.
DOCUMENT TYPE: General Review; Journal
LANGUAGE: English
REFERENCE COUNT: 125

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB One of the striking morphological features of renal failure is an increase of cell volume. This review explores the role of cell volume regulatory mechanisms in the pathophysiology of progressive renal disease. The case is made that TGF-beta, a major cytokine involved in the development of progressive renal failure, upregulates the transcription of the serum and glucocorticoid-dependent **kinase** hSGK1,involved in cell volume regulation. Excessive extracellular glucose concentrations stimulate TGF-beta1 expression and thus similarly enhance hSGK1-transcription. The **kinase** stimulates two mechanisms important for cell volume regulation, i.e. the renal epithelial Na+ channel ENaC and the thick ascending limb Na+,K+,2Cl(-) cotransporter BSC1. On the one hand, stimulation of renal tubular transport leads to renal retention of Na+, which favours the development of hypertension. On the other, the increase of cell volume stimulates protein synthesis and inhibits protein degradation, contributing to the enhanced net formation and deposition of matrix proteins. At later stages, the increase of cell volume may be reversed to atrophy, and cell death may lead to loss of functional tissue. In conclusion, progressive renal disease is paralleled by deranged cell volume regulatory mechanisms.

L7 ANSWER 19 OF 20 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 97:351584 SCISEARCH
THE GENUINE ARTICLE: WV421
TITLE: **h-sgk**, a novel human serine threonine protein **kinase**, is transcriptionally controlled by cell volume
AUTHOR: Waldegger S (Reprint); Raber G; Sailer E; Barth P; Lang F
CORPORATE SOURCE: UNIV TUBINGEN, D-72076 TUBINGEN, GERMANY
COUNTRY OF AUTHOR: GERMANY
SOURCE: PFLUGERS ARCHIV-EUROPEAN JOURNAL OF PHYSIOLOGY, (NOV-DEC 1997) Vol. 433, No. 6, Supp. [S], pp. P357-P357.
Publisher: SPRINGER VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010.
ISSN: 0031-6768.
DOCUMENT TYPE: Conference; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 0

L7 ANSWER 20 OF 20 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:756527 HCAPLUS
DOCUMENT NUMBER: 133:325643
TITLE: Antifibrotic formulations containing inhibitors of cell-volume-regulated human **kinase h -sgk**
INVENTOR(S): Lang, Florian; Waldegger, Siegfried; Wagner, Carsten; Broer, Stefan; Klingel, Karin

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PATENT ASSIGNEE(S):      Germany
SOURCE:                  PCT Int. Appl., 32 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                German
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
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     WO 2000062781      A1   20001026       WO 2000-EP3578   20000419
         W:  AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ,
             DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS,
             JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK,
             MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ,
             TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ,
             MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
             DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
             CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     DE 19917990        A1   20001102       DE 1999-19917990 19990420
     BR 2000009914      A    20020108       BR 2000-9914     20000419
     EP 1171131         A1   20020116       EP 2000-922655   20000419
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO
     JP 2002542196      T2   20021210       JP 2000-611917   20000419
     NO 2001005054      A    20011214       NO 2001-5054     20011017
     ZA 2001008610      A    20020102       ZA 2001-8610     20011019
PRIORITY APPLN. INFO.:                   DE 1999-19917990 A  19990420
                                         WO 2000-EP3578   W  20000419
AB   The invention relates to medicaments which contain inhibitors or
     activators of cell-volume-regulated human serum and glucocorticoid-dependent
     kinase h-sgk, a serine-threonine
     kinase. Medicaments of this type containing staurosporin or
     chelerythrine are suitable for treating conditions, such as fibrosis, in
     which an increased or reduced expression of h-sgk is
     identified.
REFERENCE COUNT:          17    THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> s clon? or express? or recombinant
   5 FILES SEARCHED...
L8     6092312 CLON? OR EXPRESS? OR RECOMBINANT

=> d his

     (FILE 'HOME' ENTERED AT 09:30:06 ON 28 MAY 2004)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS'
     ENTERED AT 09:30:37 ON 28 MAY 2004
L1           881 S VOLUME(A)REGULATED
L2           157 S L1 AND KINASE?
L3            49 S HUMAN AND L2
L4            19 DUP REM L3 (30 DUPLICATES REMOVED)
L5            39 S "H-SGK"
L6            20 DUP REM L5 (19 DUPLICATES REMOVED)
L7            20 S L6 AND KINASE?
L8       6092312 S CLON? OR EXPRESS? OR RECOMBINANT

=> s l3 or l5
L9            73 L3 OR L5

=> s l9 and l8
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L10 49 L9 AND L8

=> dup rem l10
PROCESSING COMPLETED FOR L10
L11 22 DUP REM L10 (27 DUPLICATES REMOVED)

=> d 1-22 ibib

L11 ANSWER 1 OF 22 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2003:198707 SCISEARCH
THE GENUINE ARTICLE: 648BF
TITLE: **Expression** of the serum- and glueocorticoid-inducible protein kinase, Sgk, is a cell survival response to multiple types of environmental stress stimuli in mammary epithelial cells
AUTHOR: Leong M L L; Maiyar A C; Kim B; O'Keeffe B A; Firestone G L (Reprint)
CORPORATE SOURCE: Univ Calif Berkeley, Dept Mol & Cell Biol, 591 LSA, Berkeley, CA 94720 USA (Reprint); Univ Calif Berkeley, Dept Mol & Cell Biol, Berkeley, CA 94720 USA; Univ Calif Berkeley, Canc Res Lab, Berkeley, CA 94720 USA
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (21 FEB 2003) Vol. 278, No. 8, pp. 5871-5882.
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3996 USA.
ISSN: 0021-9258.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 86
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L11 ANSWER 2 OF 22 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2003:401550 SCISEARCH
THE GENUINE ARTICLE: 674VM
TITLE: Stimulus-dependent regulation of serum and glucocorticoid inducible protein kinase (SGK) transcription, subcellular localization and enzymatic activity
AUTHOR: Firestone G L (Reprint); Giampaolo J R; O'Keeffe B A
CORPORATE SOURCE: Univ Calif Berkeley, Dept Mol & Cell Biol, 591 LSA, Berkeley, CA 94720 USA (Reprint); Univ Calif Berkeley, Dept Mol & Cell Biol, Berkeley, CA 94720 USA; Univ Calif Berkeley, Canc Res Lab, Berkeley, CA 94720 USA
COUNTRY OF AUTHOR: USA
SOURCE: CELLULAR PHYSIOLOGY AND BIOCHEMISTRY, (APR 2003) Vol. 13, No. 1, pp. 1-12.
Publisher: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.
ISSN: 1015-8987.
DOCUMENT TYPE: General Review; Journal
LANGUAGE: English
REFERENCE COUNT: 79
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L11 ANSWER 3 OF 22 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2002:246823 SCISEARCH
THE GENUINE ARTICLE: 530ND
TITLE: Cerebral localization and regulation of the cell volume-sensitive serum- and glucocorticoid-dependent kinase SGK1
AUTHOR: Warntges S; Friedrich B; Henke G; Duranton C; Lang P A; Waldegger S; Meyermann R; Kuhl D; Speckmann E J; Obermuller N; Witzgall R; Mack A F; Wagner H J; Wagner C A; Broer S; Lang F (Reprint)

CORPORATE SOURCE: Univ Tubingen, Inst Physiol, Gmelinstr 5, D-72076 Tubingen, Germany (Reprint); Univ Tubingen, Inst Physiol, D-72076 Tubingen, Germany; Univ Tubingen, Dept Brain Res, D-72076 Tubingen, Germany; Univ Hamburg, Zentrum Mol Neurobiol, Hamburg, Germany; Univ Munster, Dept Physiol, D-4400 Munster, Germany; Univ Heidelberg, Dept Anat, D-6900 Heidelberg, Germany; Univ Tubingen, Dept Anat, D-72076 Tubingen, Germany; Yale Univ, Dept Cellular & Mol Physiol, New Haven, CT USA
COUNTRY OF AUTHOR: Germany; USA
SOURCE: PFLUGERS ARCHIV-EUROPEAN JOURNAL OF PHYSIOLOGY, (FEB 2002) Vol. 443, No. 4, pp. 617-624.
Publisher: SPRINGER-VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010 USA.
ISSN: 0031-6768.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 42
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L11 ANSWER 4 OF 22 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2003:241577 SCISEARCH
THE GENUINE ARTICLE: 654HJ
TITLE: Activation of Na+/K+-ATPase by the serum and glucocorticoid-dependent kinase isoforms
AUTHOR: Henke G; Setiawan I; Bohmer C; Lang F (Reprint)
CORPORATE SOURCE: Univ Tubingen, Inst Physiol, Gmelinstr 5, D-72076 Tubingen, Germany (Reprint); Univ Tubingen, Inst Physiol, D-72076 Tubingen, Germany
COUNTRY OF AUTHOR: Germany
SOURCE: KIDNEY & BLOOD PRESSURE RESEARCH, (DEC 2002) Vol. 25, No. 6, pp. 370-374.
Publisher: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.
ISSN: 1420-4096.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 49
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L11 ANSWER 5 OF 22 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2002312565 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12055079
TITLE: RhoA exerts a permissive effect on **volume-regulated** anion channels in vascular endothelial cells.
AUTHOR: Carton Iris; Trouet Dominique; Hermans Diane; Barth Holger; Aktories Klaus; Droogmans Guy; Jorgensen Nanna K; Hoffmann Else K; Nilius Bernd; Eggermont Jan
CORPORATE SOURCE: Laboratory of Physiology, Katholieke Universiteit Leuven, Campus Gasthuisberg, B-3000 Leuven, Belgium.
SOURCE: American journal of physiology. Cell physiology, (2002 Jul) 283 (1) C115-25.
Journal code: 100901225. ISSN: 0363-6143.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200207
ENTRY DATE: Entered STN: 20020611
Last Updated on STN: 20020717
Entered Medline: 20020716

L11 ANSWER 6 OF 22 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN

ACCESSION NUMBER: 2002:289276 SCISEARCH
THE GENUINE ARTICLE: 536XJ
TITLE: **Expression** of the serine/threonine kinase hSGK1 in chronic viral hepatitis
AUTHOR: Fillon S; Klingel K; Warntges S; Sauter M; Gabrysch S; Pestel S; Tanneur V; Waldegger S; Zipfel A; Viebahn R; Broer S; Kandolf R; Lang F (Reprint)
CORPORATE SOURCE: Univ Tubingen, Inst Physiol, Dept Physiol, Gmelinstr 5, D-72076 Tubingen, Germany (Reprint); Univ Tubingen, Inst Physiol, Dept Physiol, D-72076 Tubingen, Germany; Univ Tubingen, Dept Mol Pathol, D-72076 Tubingen, Germany; Univ Dusseldorf, Dept Internal Med, D-4000 Dusseldorf, Germany; Univ Tubingen, Dept Surg, D-72076 Tubingen, Germany; Australian Natl Univ, Sch Biochem & Mol Biol, Canberra, ACT, Australia
COUNTRY OF AUTHOR: Germany; Australia
SOURCE: CELLULAR PHYSIOLOGY AND BIOCHEMISTRY, (DEC 2002) Vol. 12, No. 1, pp. 47-54.
Publisher: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.
ISSN: 1015-8987.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 37
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L11 ANSWER 7 OF 22 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:74113 BIOSIS
DOCUMENT NUMBER: PREV200200074113
TITLE: Cell **volume-regulated human kinase h-sgk**.
AUTHOR(S): Lang, Florian [Inventor, Reprint author]; Waldegger, Siegfried [Inventor]
CORPORATE SOURCE: Im Rotbad 52, 72076 Tubingen, Germany
PATENT INFORMATION: US 6326181 December 04, 2001
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Dec. 4, 2001) Vol. 1253, No. 1. ftp://ftp.uspto.gov/pub/patdata/. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jan 2002
Last Updated on STN: 25 Feb 2002

L11 ANSWER 8 OF 22 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2002179776 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11913450
TITLE: Serum- and glucocorticoid-dependent **kinase**, cell volume, and the regulation of epithelial transport.
AUTHOR: Fillon S; Warntges S; Matskevitch J; Moschen I; Setiawan I; Gamper N; Feng Y X; Stegen C; Friedrich B; Waldegger S; Broer S; Wagner C A; Huber S M; Klingel K; Vereninov A; Lang F
CORPORATE SOURCE: Department of Physiology, University of Tubingen, Germany.
SOURCE: Comparative biochemistry and physiology. Part A, Molecular & integrative physiology, (2001 Oct) 130 (3) 367-76. Ref: 99
Journal code: 9806096. ISSN: 1095-6433.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20020401
Last Updated on STN: 20020614
Entered Medline: 20020418

L11 ANSWER 9 OF 22 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2001:922107 SCISEARCH
THE GENUINE ARTICLE: 490MJ
TITLE: Cell volume regulatory mechanisms in progression of renal disease
AUTHOR: Warntges S; Grone H J; Capasso G; Lang F (Reprint)
CORPORATE SOURCE: Univ Tubingen, Inst Physiol, Gmelinstr 5, D-76072 Tubingen, Germany (Reprint); Univ Tubingen, Dept Physiol, Tubingen, Germany
COUNTRY OF AUTHOR: Germany
SOURCE: JOURNAL OF NEPHROLOGY, (SEP-OCT 2001) Vol. 14, No. 5, pp. 319-326.
Publisher: WICHTIG EDITORE, 72/74 VIA FRIULI, 20135 MILAN, ITALY.
ISSN: 1121-8428.
DOCUMENT TYPE: General Review; Journal
LANGUAGE: English
REFERENCE COUNT: 125
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L11 ANSWER 10 OF 22 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:244741 BIOSIS
DOCUMENT NUMBER: PREV200100244741
TITLE: All three isoforms of human serum and glucocorticoid dependent kinase (**h-SGK**) upregulate voltage-gated potassium channels endogenously **expressed** in HEK293 cells.
AUTHOR(S): Fillon, S. [Reprint author]; Gamper, N. [Reprint author]; Huber, S. M. [Reprint author]; Feng, Y. X. [Reprint author]; Friedrich, B. [Reprint author]; Kobayashi, T.; Cohen, P.; Lang, F. [Reprint author]
CORPORATE SOURCE: Institute of Physiology, University of Tuebingen, Tuebingen, Germany
SOURCE: Pfluegers Archiv European Journal of Physiology, (2001) Vol. 441, No. 6 Supplement, pp. R182. print.
Meeting Info.: Joint Congress of the Scandinavian and the German Physiological Societies. Berlin, Germany. March 10-13, 2001.
CODEN: PFLABK. ISSN: 0031-6768.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 May 2001
Last Updated on STN: 19 Feb 2002

L11 ANSWER 11 OF 22 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:358201 BIOSIS
DOCUMENT NUMBER: PREV200100358201
TITLE: Association between inflammation and **expression** of human serine threonine kinase (**h-sgk**) in fetal and neonatal lung tissue.
AUTHOR(S): Speer, Christian P. [Reprint author]; Schmidt, Beate [Reprint author]; Cao, Lei [Reprint author]; Klingel, Karin; Mackensen-Haen, Susanne; Lang, Florian
CORPORATE SOURCE: University Children's Hospital, Wuerzburg, Germany
SOURCE: Biology of the Neonate, (May, 2001) Vol. 80, No. Supplement 1, pp. 35. print.
Meeting Info.: Proceedings of the 16th International

Workshop on Surfactant Replacement. Edinburgh, Scotland. June 02-04, 2001.
CODEN: BNEOBV. ISSN: 0006-3126.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Aug 2001
Last Updated on STN: 19 Feb 2002

L11 ANSWER 12 OF 22 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:756527 HCAPLUS
DOCUMENT NUMBER: 133:325643
TITLE: Antifibrotic formulations containing inhibitors of cell-**volume-regulated human kinase h-sgk**
INVENTOR(S): Lang, Florian; Waldegger, Siegfried; Wagner, Carsten; Broer, Stefan; Klingel, Karin
PATENT ASSIGNEE(S): Germany
SOURCE: PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000062781	A1	20001026	WO 2000-EP3578	20000419
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
DE 19917990	A1	20001102	DE 1999-19917990	19990420
BR 2000009914	A	20020108	BR 2000-9914	20000419
EP 1171131	A1	20020116	EP 2000-922655	20000419
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002542196	T2	20021210	JP 2000-611917	20000419
NO 2001005054	A	20011214	NO 2001-5054	20011017
ZA 2001008610	A	20020102	ZA 2001-8610	20011019

PRIORITY APPLN. INFO.: DE 1999-19917990 A 19990420
WO 2000-EP3578 W 20000419
REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 13 OF 22 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2001034894 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11052997
TITLE: **Expression** of cell **volume-regulated kinase h-sgk** in pancreatic tissue.
AUTHOR: Klingel K; Warntges S; Bock J; Wagner C A; Sauter M; Waldegger S; Kandolf R; Lang F
CORPORATE SOURCE: Department of Molecular Pathology, Institute of Pathology, University of Tubingen, D-72076, Tubingen, Germany.
SOURCE: American journal of physiology. Gastrointestinal and liver physiology, (2000 Nov) 279 (5) G998-G1002.
Journal code: 100901227. ISSN: 0193-1857.
PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20020420
Entered Medline: 20001130

L11 ANSWER 14 OF 22 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 4
ACCESSION NUMBER: 2001126681 EMBASE
TITLE: **Expression** and localization of serum/glucocorticoid-induced kinase in the rat ovary: Relation to follicular growth and differentiation.
AUTHOR: Alliston T.N.; Gonzalez-Robayna I.J.; Buse P.; Firestone G.L.; Richards J.S.
CORPORATE SOURCE: Dr. J.S. Richards, Department of Molecular Biology, Baylor College of Medicine, Houston, TX 77030, United States. joanner@bcm.tmc.edu
SOURCE: Endocrinology, (2000) 141/1 (385-395).
Refs: 54
ISSN: 0013-7227 CODEN: ENDOAO
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
010 Obstetrics and Gynecology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L11 ANSWER 15 OF 22 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 2001067208 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11093030
TITLE: **h-sgk** serine-threonine protein **kinase** as transcriptional target of p38/MAP **kinase** pathway in HepG2 **human** hepatoma cells.
AUTHOR: Waldegger S; Gabrysch S; Barth P; Fillon S; Lang F
CORPORATE SOURCE: Institut fur Physiologie I, Gmelinstr. 5, D-72076 Tubingen, Germany.
SOURCE: Cellular physiology and biochemistry : international journal of experimental cellular physiology, biochemistry, and pharmacology, (2000) 10 (4) 203-8.
Journal code: 9113221. ISSN: 1015-8987.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20020420
Entered Medline: 20001222

L11 ANSWER 16 OF 22 MEDLINE on STN
ACCESSION NUMBER: 2001067206 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11093028
TITLE: The shrinkage-activated Na(+) conductance of rat hepatocytes and its possible correlation to rENaC.
AUTHOR: Bohmer C; Wagner C A; Beck S; Moschen I; Melzig J; Werner A; Lin J T; Lang F; Wehner F
CORPORATE SOURCE: Max-Planck-Institut fur molekulare Physiologie, Abteilung Epithelphysiologie, Otto-Hahn-Str. 11, 44227 Dortmund, Germany.
SOURCE: Cellular physiology and biochemistry : international

journal of experimental cellular physiology, biochemistry, and pharmacology, (2000) 10 (4) 187-94.
Journal code: 9113221. ISSN: 1015-8987.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20020420
Entered Medline: 20001222

L11 ANSWER 17 OF 22 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 1999238882 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10220500
TITLE: **h-sgk** serine-threonine protein kinase gene as transcriptional target of transforming growth factor beta in human intestine.
AUTHOR: Waldegger S; Klingel K; Barth P; Sauter M; Rfer M L; Kandolf R; Lang F
CORPORATE SOURCE: Institute of Physiology, University of Tubingen, Tubingen, Germany.. florian.lang@uni-tuebingen.de
SOURCE: Gastroenterology, (1999 May) 116 (5) 1081-8.
Journal code: 0374630. ISSN: 0016-5085.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990618
Last Updated on STN: 20020420
Entered Medline: 19990607

L11 ANSWER 18 OF 22 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 1999:888633 SCISEARCH
THE GENUINE ARTICLE: 255JC
TITLE: Osmotic cell swelling-induced ATP release mediates the activation of extracellular signal-regulated protein **kinase** (Erk)-1/2 but not the activation of osmo-sensitive anion channels
AUTHOR: vanderWijk T (Reprint); deJonge H R; Tilly B C
CORPORATE SOURCE: ERASMUS UNIV, DEPT BIOCHEM, CARDIOVASC RES INST COEUR, FAC MED & HLTH SCI, POB 1738, NL-3000 DR ROTTERDAM, NETHERLANDS (Reprint)
COUNTRY OF AUTHOR: NETHERLANDS
SOURCE: BIOCHEMICAL JOURNAL, (1 NOV 1999) Vol. 343, Part 3, pp. 579-586.
Publisher: PORTLAND PRESS, 59 PORTLAND PLACE, LONDON W1N 3AJ, ENGLAND.
ISSN: 0264-6021.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 48
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L11 ANSWER 19 OF 22 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
ACCESSION NUMBER: 1998-10366 BIOTECHDS
TITLE: New nucleic acid encoding cell-volume regulating kinase **h-sgk** and related proteins;
enzyme and protein used for diagnosis and therapy of condition related to cell-volume change
AUTHOR: Lang F; Waldegger S
PATENT ASSIGNEE: Dade-Behring-Marburg

LOCATION: Marburg, Germany.
PATENT INFO: EP 861896 2 Sep 1998
APPLICATION INFO: EP 1998-101338 27 Jan 1998
PRIORITY INFO: DE 1997-1008173 28 Feb 1997
DOCUMENT TYPE: Patent
LANGUAGE: German
OTHER SOURCE: WPI: 1998-449109 [39]

L11 ANSWER 20 OF 22 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 8
ACCESSION NUMBER: 1998305122 EMBASE
TITLE: **Cloning** of sgk serine-threonine protein **kinase** from shark rectal gland - A gene induced by hypertonicity and secretagogues.
AUTHOR: Waldegger S.; Barth P.; Forrest J.N. Jr.; Greger R.; Lang F.
CORPORATE SOURCE: S. Waldegger, Department of Physiology 1, University of Tubingen, Gmelinstr. 5, D-72076 Tubingen, Germany
SOURCE: Pflugers Archiv European Journal of Physiology, (1998) 436/4 (575-580).
Refs: 35
ISSN: 0031-6768 CODEN: PFLABK
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L11 ANSWER 21 OF 22 MEDLINE on STN DUPLICATE 9
ACCESSION NUMBER: 97272242 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9114008
TITLE: **Cloning** and characterization of a putative human serine/threonine protein kinase transcriptionally modified during anisotonic and isotonic alterations of cell volume.
AUTHOR: Waldegger S; Barth P; Raber G; Lang F
CORPORATE SOURCE: Physiologisches Institut I der Eberhard-Karls-Universitat, D-72076 Tubingen, Germany.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1997 Apr 29) 94 (9) 4440-5.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-Y10032
ENTRY MONTH: 199705
ENTRY DATE: Entered STN: 19970609
Last Updated on STN: 20020420
Entered Medline: 19970527

L11 ANSWER 22 OF 22 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 95:115826 SCISEARCH
THE GENUINE ARTICLE: QE655
TITLE: VOLUME-ACTIVATED CHLORIDE CURRENTS ASSOCIATED WITH THE MULTIDRUG-RESISTANCE P-GLYCOPROTEIN
AUTHOR: HIGGINS C F (Reprint)
CORPORATE SOURCE: UNIV OXFORD, JOHN RADCLIFFE HOSP, NUFFIELD DEPT CLIN BIOCHEM, OXFORD OX3 9DU, ENGLAND (Reprint); UNIV OXFORD, JOHN RADCLIFFE HOSP, INST MOLEC MED, OXFORD OX3 9DU, ENGLAND
COUNTRY OF AUTHOR: ENGLAND
SOURCE: JOURNAL OF PHYSIOLOGY-LONDON, (JAN 1995) Vol. 482P, pp. S31-S36.

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                    ISSN: 0022-3751.
DOCUMENT TYPE:      Article; Journal
FILE SEGMENT:       LIFE
LANGUAGE:           ENGLISH
REFERENCE COUNT:    40
                   *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*


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     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS'
     ENTERED AT 09:30:37 ON 28 MAY 2004
L1          881 S VOLUME(A)REGULATED
L2          157 S L1 AND KINASE?
L3           49 S HUMAN AND L2
L4           19 DUP REM L3 (30 DUPLICATES REMOVED)
L5           39 S "H-SGK"
L6           20 DUP REM L5 (19 DUPLICATES REMOVED)
L7           20 S L6 AND KINASE?
L8      6092312 S CLON? OR EXPRESS? OR RECOMBINANT
L9           73 S L3 OR L5
L10          49 S L9 AND L8
L11          22 DUP REM L10 (27 DUPLICATES REMOVED)

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E2            2     LANG EVERETT H/AU
E3         3108 --> LANG F/AU
E4           62     LANG F A/AU
E5            1     LANG F B/AU
E6           29     LANG F C/AU
E7            7     LANG F D/AU
E8          144     LANG F F/AU
E9            2     LANG F F JR/AU
E10           2     LANG F G/AU
E11          34     LANG F H/AU
E12          38     LANG F J/AU

=> s e3
L12        3108 "LANG F"/AU

=> e waldegger s/au
E1            4     WALDEGGER P/AU
E2            7     WALDEGGER PETRA/AU
E3          348 --> WALDEGGER S/AU
E4            1     WALDEGGER SIEGFREID/AU
E5            2     WALDEGGER SIEGFRID/AU
E6          132     WALDEGGER SIEGFRIED/AU
E7            2     WALDEGGER SIEGRIED/AU
E8            1     WALDEGGER W/AU
E9            1     WALDEGRAVE/AU
E10           2     WALDEGRAVE C/AU
E11           2     WALDEGRAVE M/AU
E12          18     WALDEGRAVE W/AU

=> s e3
L13         348 "WALDEGGER S"/AU

=> s l12 or l13
L14        3155 L12 OR L13

=> d his
```

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     (FILE 'HOME' ENTERED AT 09:30:06 ON 28 MAY 2004)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS'
     ENTERED AT 09:30:37 ON 28 MAY 2004
L1           881 S VOLUME(A)REGULATED
L2           157 S L1 AND KINASE?
L3            49 S HUMAN AND L2
L4            19 DUP REM L3 (30 DUPLICATES REMOVED)
L5            39 S "H-SGK"
L6            20 DUP REM L5 (19 DUPLICATES REMOVED)
L7            20 S L6 AND KINASE?
L8       6092312 S CLON? OR EXPRESS? OR RECOMBINANT
L9            73 S L3 OR L5
L10           49 S L9 AND L8
L11           22 DUP REM L10 (27 DUPLICATES REMOVED)
                 E LANG F/AU
L12         3108 S E3
                 E WALDEGGER S/AU
L13          348 S E3
L14         3155 S L12 OR L13

=> s l9 and l14
L15           30 L9 AND L14

=> dup rem l15
PROCESSING COMPLETED FOR L15
L16           15 DUP REM L15 (15 DUPLICATES REMOVED)

=> d 1-15 ibib ab

L16  ANSWER 1 OF 15  SCISEARCH  COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER:    2004:390716  SCISEARCH
THE GENUINE ARTICLE: 814KP
TITLE:               Association of the serum and glucocorticoid regulated
                     kinase (sgk1) gene with QT interval
AUTHOR:              Busjahn A; Seebohm G; Maier G; Toliat M R; Nurnberg P;
                     Aydin A; Luft F C (Reprint); Lang F
CORPORATE SOURCE:    HELIOS Kliniken Berlin, Franz Volhard Clin, Wiltberg Str
                     50, D-13125 Berlin, Germany (Reprint); HELIOS Kliniken
                     Berlin, Franz Volhard Clin, D-13125 Berlin, Germany;
                     Humboldt Univ, Fac Med Charite, Max Delbruck Ctr Mol Med,
                     Berlin, Germany; Univ Tubingen, Dept Physiol, D-72074
                     Tubingen, Germany; Max Delbruck Ctr Mol Med, Gene Mapping
                     Ctr, Berlin, Germany
COUNTRY OF AUTHOR:   Germany
SOURCE:              CELLULAR PHYSIOLOGY AND BIOCHEMISTRY, (APR 2004) Vol. 14,
                     No. 3, pp. 135-142.
                     Publisher: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL,
                     SWITZERLAND.
                     ISSN: 1015-8987.
DOCUMENT TYPE:       Article; Journal
LANGUAGE:            English
REFERENCE COUNT:     65
                    *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*
AB       The serum and glucocorticoid inducible kinase (SGK1) is well known to
     up-regulate the renal epithelial Na+ channel ENaC. Excessive SGK1 activity
     would be expected to cause renal Na+ retention and blood pressure
     increase. Certain polymorphisms of the SGK1 gene (E8CC/CT; 16CC) are
     indeed associated with moderately enhanced blood pressure. We have
     recently disclosed another function of SGK1, i.e. the stimulation of the
     slowly activating K+ channel KCNE1/KCNQ1. Among the functions of this
     channel is the repolarisation of cardiac myocytes. Accordingly, defective
     KCNE1 and/or KCNQ1 lead to long QT syndrome, a disorder causing fainting
```

and sudden cardiac death. In the present study we demonstrate that coexpression of SGK1 in Xenopus oocytes increases KCNQ1/KCNE1 induced current without significantly altering voltage dependence, activation and deactivation kinetics. To test for the relevance of SGK1 in human cardiac repolarization, we analysed the ECG of monozygotic (MZ) (126 pairs) and dizygotic (DZ) (70 pairs) twin subjects and parents of DZ twins. The E8CC/CT;16CC polymorphism was indeed significantly ($p < 0.025$) associated with shortened age and gender corrected QT interval. No significant differences were observed in any other ECG parameter, including heart rate, P, PQ and QRS. We conclude that the regulation of KCNE1/KCNQ1 by SGK1 is similarly relevant for the repolarization of cardiac myocytes as for regulation of renal ENaC activity and blood pressure control.

L16 ANSWER 2 OF 15 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2002:246823 SCISEARCH
THE GENUINE ARTICLE: 530ND
TITLE: Cerebral localization and regulation of the cell volume-sensitive serum- and glucocorticoid-dependent kinase SGK1
AUTHOR: Warntges S; Friedrich B; Henke G; Duranton C; Lang P A; **Waldegger S**; Meyermann R; Kuhl D; Speckmann E J; Obermuller N; Witzgall R; Mack A F; Wagner H J; Wagner C A; Broer S; **Lang F (Reprint)**
CORPORATE SOURCE: Univ Tubingen, Inst Physiol, Gmelinstr 5, D-72076 Tubingen, Germany (Reprint); Univ Tubingen, Inst Physiol, D-72076 Tubingen, Germany; Univ Tubingen, Dept Brain Res, D-72076 Tubingen, Germany; Univ Hamburg, Zentrum Mol Neurobiol, Hamburg, Germany; Univ Munster, Dept Physiol, D-4400 Munster, Germany; Univ Heidelberg, Dept Anat, D-6900 Heidelberg, Germany; Univ Tubingen, Dept Anat, D-72076 Tubingen, Germany; Yale Univ, Dept Cellular & Mol Physiol, New Haven, CT USA
COUNTRY OF AUTHOR: Germany; USA
SOURCE: PFLUGERS ARCHIV-EUROPEAN JOURNAL OF PHYSIOLOGY, (FEB 2002) Vol. 443, No. 4, pp. 617-624.
Publisher: SPRINGER-VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010 USA.
ISSN: 0031-6768.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 42

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The serum- and glucocorticoid-dependent kinase SGK1 is regulated by alterations of cell volume, whereby cell shrinkage increases and cell swelling decreases the transcription, expression and activity of SGK1. The kinase is expressed in all human tissues studied including the brain. The present study was performed to localize the sites of SGK1 transcription in the brain, to elucidate the influence of the hydration status on SGK1 transcription and to explore the functional significance of altered SGK1 expression. Northern blot analysis of human brain showed SGK1 to be expressed in all cerebral structures examined: amygdala, caudate nucleus, corpus callosum, hippocampus, substantia nigra, subthalamic nucleus and thalamus. In situ hybridization and immunohistochemistry in the rat revealed increased expression of SGK1 in neurons of the hippocampal area CA3 after dehydration, compared with similar slices from brains of euvolaemic rats. Additionally, several oligodendrocytes, a few microglial cells, but no astrocytes, were positive for SGK1. The abundance of SGK1 mRNA in the temporal lobe, including hippocampus, was increased by dehydration and SGK1 transcription in neuroblastoma cells was stimulated by an increase of extracellular osmolarity. Co-expression studies in Xenopus laevis oocytes revealed that SGK1 markedly increased the activity of the neuronal K+ channel Kv1.3. As activation of K+ channels modifies excitation of neuronal cells, SGK1 may participate in the regulation of

neuronal excitability.

L16 ANSWER 3 OF 15 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2003:241577 SCISEARCH
THE GENUINE ARTICLE: 654HJ
TITLE: Activation of Na+/K+-ATPase by the serum and glucocorticoid-dependent kinase isoforms
AUTHOR: Henke G; Setiawan I; Bohmer C; **Lang F (Reprint)**
CORPORATE SOURCE: Univ Tubingen, Inst Physiol, Gmelinstr 5, D-72076 Tubingen, Germany (Reprint); Univ Tubingen, Inst Physiol, D-72076 Tubingen, Germany
COUNTRY OF AUTHOR: Germany
SOURCE: KIDNEY & BLOOD PRESSURE RESEARCH, (DEC 2002) Vol. 25, No. 6, pp. 370-374.
Publisher: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.
ISSN: 1420-4096.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 49
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Background/Aim: Expression of the constitutively active form of serum and glucocorticoid-dependent kinase ((S422D)SGK1) in Xenopus oocytes has recently been shown to upregulate endogenous Na+/K+-ATPase activity, an effect presumably participating in the regulation of cellular K+ uptake and transepithelial Na+ transport. SGK1 and the two isoforms SGK2 and SGK3 are stimulated by insulin and insulin-like growth factor-1 (IGF-1), which have been shown to enhance Na+/K+-ATPase activity in a variety of cells. The present experiments have been performed to elucidate whether or not wild-type SGK1, SGK2 and SGK3 are similar to (S422D)SGK1 in being effective regulators of Na+/K+-ATPase. Methods: To this end, dual-electrode voltage clamp experiments were performed in Xenopus oocytes injected either with water or with mRNA of constitutively active (S422D)SGK1 and wild-type SGK1, SGK2 or SGK3. Na+/K+-ATPase activity was estimated from the outward-directed current created by readdition of extracellular K+ in the presence of K+ channel blocker Ba2+ following a 10-min exposure to K+-free extracellular fluid. Results: The outward-directed current was fully abolished by incubation with 1 mM ouabain and was significantly larger in oocytes expressing (S422D)SGK1, SGK1, SGK2 or SGK3, as compared to those injected with water. Conclusion: The stimulating effect of SGK1 on the Xenopus oocyte Na+/K+-ATPase is mimicked by the isoforms SGK2 and SGK3. Thus, all three kinases may participate in the regulation of Na+/K+-ATPase activity by hormones such as insulin and IGF-1. Copyright (C) 2002 S. Karger AG, Basel.

L16 ANSWER 4 OF 15 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2002:289276 SCISEARCH
THE GENUINE ARTICLE: 536XJ
TITLE: Expression of the serine/threonine kinase hSGK1 in chronic viral hepatitis
AUTHOR: Fillon S; Klingel K; Warntges S; Sauter M; Gabrysch S; Pestel S; Tanneur V; **Waldegger S**; Zipfel A; Viebahn R; Broer S; Kandolf R; **Lang F (Reprint)**
CORPORATE SOURCE: Univ Tubingen, Inst Physiol, Dept Physiol, Gmelinstr 5, D-72076 Tubingen, Germany (Reprint); Univ Tubingen, Inst Physiol, Dept Physiol, D-72076 Tubingen, Germany; Univ Tubingen, Dept Mol Pathol, D-72076 Tubingen, Germany; Univ Dusseldorf, Dept Internal Med, D-4000 Dusseldorf, Germany; Univ Tubingen, Dept Surg, D-72076 Tubingen, Germany; Australian Natl Univ, Sch Biochem & Mol Biol, Canberra, ACT, Australia
COUNTRY OF AUTHOR: Germany; Australia
SOURCE: CELLULAR PHYSIOLOGY AND BIOCHEMISTRY, (DEC 2002) Vol. 12, No. 1, pp. 47-54.

Publisher: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.
ISSN: 1015-8987.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 37
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The human serine/threonine kinase hSGK1 is expressed ubiquitously with highest transcript levels in pancreas and liver. This study has been performed to determine the hSGK1 distribution in normal liver and its putative role in fibrosing liver disease. HSGK1-localization was determined by in situ hybridization, regulation of hSGK1-transcription by Northern blotting, fibronectin synthesis and hSGK1 phosphorylation by Western blotting. In normal liver hSGK1 was mainly transcribed by Kupffer cells. In liver tissue from patients with chronic viral hepatitis, hSGK1 transcript levels were excessively high in numerous activated Kupffer cells and inflammatory cells localized within fibrous septum formations. HSGK1 transcripts were also detected in activated hepatic stellate cells. Accordingly, Western blotting revealed that tissue from fibrotic liver expresses excessive hSGK1 protein as compared to normal liver. TGF-beta1 (2 ng/ml) increases hSGK1 transcription in both human U937 macrophages and HepG2 hepatoma cells. H2O2 (0.3 mM) activated hSGK1 and increased fibronectin formation in HepG2 cells overexpressing hSGK1 but not in HepG2 cells expressing the inactive mutant hSGK1(K127R). In conclusion hSGK1 is upregulated by TGF-beta1 during hepatitis and may contribute to enhanced matrix formation during fibrosing liver disease. Copyright (C) 2002 S. Karger AG, Basel.

L16 ANSWER 5 OF 15 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2002179776 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11913450
TITLE: Serum- and glucocorticoid-dependent **kinase**, cell volume, and the regulation of epithelial transport.
AUTHOR: Fillon S; Warntges S; Matskevitch J; Moschen I; Setiawan I; Gamper N; Feng Y X; Stegen C; Friedrich B; **Waldegger S**; Broer S; Wagner C A; Huber S M; Klingel K; Vereninov A; **Lang F**
CORPORATE SOURCE: Department of Physiology, University of Tubingen, Germany.
SOURCE: Comparative biochemistry and physiology. Part A, Molecular & integrative physiology, (2001 Oct) 130 (3) 367-76. Ref: 99
Journal code: 9806096. ISSN: 1095-6433.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20020401
Last Updated on STN: 20020614
Entered Medline: 20020418

AB Ample pharmacological evidence points to a role of **kinases** in the regulation of cell volume. Given the limited selectivity of most inhibitors, however, the specific molecules involved have remained largely elusive. The search for cell **volume regulated** genes in liver HepG2 cells led to the discovery of the **human** serum- and glucocorticoid-dependent serine/threonine **kinase** hsgk1. Transcription and expression of hsgk1 is markedly and rapidly upregulated by osmotic and isotonic cell shrinkage. The effect of osmotic cell shrinkage on hsgk1 is mediated by p38 **kinase**. Further stimuli of hsgk1 transcription include glucocorticoids, aldosterone, TGF-beta1, serum, increase of intracellular Ca2+ and phorbolesters, whereas cAMP downregulates hsgk1 transcription. The hsgk1 protein is expressed in

several epithelial tissues including **human** pancreas, intestine, kidney, and shark rectal gland. Co-expression of hsgk1 with the renal epithelial Na+-channel ENaC or the Na+/K+/2Cl(-)-cotransporter NKCC2 (BSC1) in Xenopus oocytes, accelerates insertion of the transport proteins into the cell membrane and thus, stimulates channel or transport activity. Thus, hsgk1 participates in the regulation of transport by steroids and secretagogues increasing intracellular Ca2+-activity. The stimulation of hsgk1 transcription by TGF-beta1 may further bear pathophysiological relevance.

L16 ANSWER 6 OF 15 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2001:922107 SCISEARCH
THE GENUINE ARTICLE: 490MJ
TITLE: Cell volume regulatory mechanisms in progression of renal disease
AUTHOR: Warntges S; Grone H J; Capasso G; **Lang F (Reprint)**
CORPORATE SOURCE: Univ Tubingen, Inst Physiol, Gmelinstr 5, D-76072 Tubingen, Germany (Reprint); Univ Tubingen, Dept Physiol, Tubingen, Germany
COUNTRY OF AUTHOR: Germany
SOURCE: JOURNAL OF NEPHROLOGY, (SEP-OCT 2001) Vol. 14, No. 5, pp. 319-326.
Publisher: WICHTIG EDITORE, 72/74 VIA FRIULI, 20135 MILAN, ITALY.
ISSN: 1121-8428.
DOCUMENT TYPE: General Review; Journal
LANGUAGE: English
REFERENCE COUNT: 125

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB One of the striking morphological features of renal failure is an increase of cell volume. This review explores the role of cell volume regulatory mechanisms in the pathophysiology of progressive renal disease. The case is made that TGF-beta, a major cytokine involved in the development of progressive renal failure, upregulates the transcription of the serum and glucocorticoid-dependent kinase hSGK1,involved in cell volume regulation. Excessive extracellular glucose concentrations stimulate TGF-beta1 expression and thus similarly enhance hSGK1-transcription. The kinase stimulates two mechanisms important for cell volume regulation, i.e. the renal epithelial Na+ channel ENaC and the thick ascending limb Na+,K+,2Cl(-) cotransporter BSC1. On the one hand, stimulation of renal tubular transport leads to renal retention of Na+, which favours the development of hypertension. On the other, the increase of cell volume stimulates protein synthesis and inhibits protein degradation, contributing to the enhanced net formation and deposition of matrix proteins. At later stages, the increase of cell volume may be reversed to atrophy, and cell death may lead to loss of functional tissue. In conclusion, progressive renal disease is paralleled by deranged cell volume regulatory mechanisms.

L16 ANSWER 7 OF 15 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:244741 BIOSIS
DOCUMENT NUMBER: PREV200100244741
TITLE: All three isoforms of human serum and glucocorticoid dependent kinase (**h-SGK**) upregulate voltage-gated potassium channels endogenously expressed in HEK293 cells.
AUTHOR(S): Fillon, S. [Reprint author]; Gamper, N. [Reprint author]; Huber, S. M. [Reprint author]; Feng, Y. X. [Reprint author]; Friedrich, B. [Reprint author]; Kobayashi, T.; Cohen, P.; **Lang, F.** [Reprint author]
CORPORATE SOURCE: Institute of Physiology, University of Tuebingen, Tuebingen, Germany
SOURCE: Pfluegers Archiv European Journal of Physiology, (2001) Vol. 441, No. 6 Supplement, pp. R182. print.

Meeting Info.: Joint Congress of the Scandinavian and the German Physiological Societies. Berlin, Germany. March 10-13, 2001.
CODEN: PFLABK. ISSN: 0031-6768.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 May 2001
Last Updated on STN: 19 Feb 2002

L16 ANSWER 8 OF 15 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2001034894 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11052997
TITLE: Expression of cell **volume-regulated kinase h-sgk** in pancreatic tissue.
AUTHOR: Klingel K; Warntges S; Bock J; Wagner C A; Sauter M; **Waldegger S**; Kandolf R; **Lang F**
CORPORATE SOURCE: Department of Molecular Pathology, Institute of Pathology, University of Tubingen, D-72076, Tubingen, Germany.
SOURCE: American journal of physiology. Gastrointestinal and liver physiology, (2000 Nov) 279 (5) G998-G1002.
Journal code: 100901227. ISSN: 0193-1857.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20020420
Entered Medline: 20001130

AB Transcript levels of the **human** serine/threonine **kinase h-sgk** have been found to be highest in pancreas. In the present study, localization and regulation of **h-sgk** transcription in pancreatic tissue were elucidated. As was apparent from radioactive in situ hybridization, most pancreatic acinar cells expressed high levels of **h-sgk** mRNA. **h-sgk** mRNA-positive cells were also found in ductal epithelia but not in pancreatic islets. In biopsy specimens from patients with pancreatitis, **h-sgk** mRNA levels were decreased in acinar cells but abundant in numerous mononuclear interstitial cells within areas of pancreatic necrosis and fibrosis. As shown by Northern blotting, **h-sgk** transcription in DAN-G pancreatic tumor cells is upregulated by osmotic cell shrinkage, serum, phorbol esters (phorbol 12,13-didecanoate), and Ca(2+) ionophore A-23187 and decreased by staurosporine and cAMP. In conclusion, **h-sgk** transcription is regulated not only by cell volume but also by serum, protein **kinase** C stimulation, cAMP, and increase of intracellular Ca(2+) activity. The **kinase** may participate not only in normal function of exocrine pancreas but also in fibrosing pancreatitis.

L16 ANSWER 9 OF 15 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2001067208 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11093030
TITLE: **h-sgk** serine-threonine protein **kinase** as transcriptional target of p38/MAP **kinase** pathway in HepG2 **human** hepatoma cells.
AUTHOR: **Waldegger S**; Gabrysch S; Barth P; Fillon S; **Lang F**
CORPORATE SOURCE: Institut fur Physiologie I, Gmelinstr. 5, D-72076 Tubingen, Germany.

SOURCE: Cellular physiology and biochemistry : international journal of experimental cellular physiology, biochemistry, and pharmacology, (2000) 10 (4) 203-8.
Journal code: 9113221. ISSN: 1015-8987.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20020420
Entered Medline: 20001222

AB The **human** serum and glucocorticoid dependent serine/threonine **kinase h-sgk** has previously been discovered as cell **volume regulated** gene. The present study has been performed to elucidate the involvement of p38-**kinase** in the transcriptional control of **h-sgk** by osmotic cell shrinkage. The p38-**kinase** has previously been cloned as the mammalian homologue of HOG1 **kinase**, which constitutes a part of the osmosensor in the yeast Saccharomyces cerevisiae. Phosphorylated (active) p38-**kinase** has been estimated with Western blotting, transcription of hsgk using Northern blotting. Both, increase of extracellular NaCl concentration by 50 mmol/l and addition of 10 micromol/l anisomycin increase phosphorylation of the p38-**kinase** within 5 to 10 minutes. **h-sgk** transcription is upregulated by addition of 50 mmol/l NaCl and by anisomycin (10 micromol/l), effects completely inhibited by the specific p38-**kinase** inhibitor, SB 203580 (10 micromol/l). In conclusion, the stimulation of **h-sgk** transcription by osmotic cell shrinkage is mediated by p38-**kinase**.

L16 ANSWER 10 OF 15 MEDLINE on STN
ACCESSION NUMBER: 2001067206 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11093028
TITLE: The shrinkage-activated Na(+) conductance of rat hepatocytes and its possible correlation to rENaC.
AUTHOR: Bohmer C; Wagner C A; Beck S; Moschen I; Melzig J; Werner A; Lin J T; **Lang F**; Wehner F
CORPORATE SOURCE: Max-Planck-Institut fur molekulare Physiologie, Abteilung Epithelphysiologie, Otto-Hahn-Str. 11, 44227 Dortmund, Germany.
SOURCE: Cellular physiology and biochemistry : international journal of experimental cellular physiology, biochemistry, and pharmacology, (2000) 10 (4) 187-94.
Journal code: 9113221. ISSN: 1015-8987.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20020420
Entered Medline: 20001222

AB At moderate cell shrinkage, activation of Na(+) channels is the most prominent mechanism of regulatory cell volume increase in rat hepatocytes. The amiloride sensitivity of these channels suggests a relation to the family of epithelial Na(+) channels (ENaCs). The present study was performed to determine the pharmacological profile of shrinkage-activated Na(+) channels and to test for ENaC expression in primary cultures of rat hepatocytes; in addition, the influence of the cell **volume regulated** serine/threonine **kinase** hSGK on activity and pharmacological profile of rENaC was examined in Xenopus oocytes. Conventional electrophysiology in hepatocytes reveals that the

shrinkage-activated Na(+) channel is inhibited by amiloride and EIPA with IC(50) values of 6.0 and 0.12 micromol/l, respectively. Western blots and RT-PCR demonstrate that rat hepatocytes do express all three subunits (alpha, beta, gamma) of ENaC. Coexpression of hSGK with rENaC in Xenopus oocytes reveals that the **kinase** stimulates ENaC by a factor of 4. Moreover, hSGK decreases the affinity to amiloride (increase of IC(50) from 0.12 to 0.26 micromol/l) and increases the affinity to EIPA (decrease of IC(50) from 250 to 50 micromol/l). In conclusion, rat hepatocytes express ENaC, which is activated by the cell volume-sensitive **kinase** hSGK. ENaC may contribute to the Na(+) channels activated by osmotic cell shrinkage in hepatocytes, whereby the relatively low amiloride and high EIPA sensitivity of the channel could at least be partially due to modification by SGK, which decreases the amiloride and increases the EIPA sensitivity of ENaC.

L16 ANSWER 11 OF 15 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 1999238882 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10220500
TITLE: **h-sgk** serine-threonine protein kinase gene as transcriptional target of transforming growth factor beta in human intestine.
AUTHOR: **Waldegger S**; Klingel K; Barth P; Sauter M; Rfer M L; Kandolf R; **Lang F**
CORPORATE SOURCE: Institute of Physiology, University of Tubingen, Tubingen, Germany.. florian.lang@uni-tuebingen.de
SOURCE: Gastroenterology, (1999 May) 116 (5) 1081-8.
Journal code: 0374630. ISSN: 0016-5085.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990618
Last Updated on STN: 20020420
Entered Medline: 19990607

AB BACKGROUND & AIMS: Recently, the immediate early gene **h-sgk** was cloned as a hypertonicity-induced gene from human hepatoma cells. The aim of this study was to localize **h-sgk** messenger RNA (mRNA) expression in normal and inflamed intestinal mucosa and to identify potential transcriptional regulators. METHODS: **h-sgk** mRNA in small intestinal mucosa from healthy persons and patients with Crohn's disease was determined by in situ hybridization. Transcriptional regulation was studied by Northern blot analysis of total RNA isolated from cultured human Intestine 407, U937, and HepG2 cells. RESULTS: In normal ileum, **h-sgk** mRNA was selectively localized to the apical villus enterocytes, whereas no staining was detected in crypt cells. In Crohn's disease, enterocytes of the crypts expressed **h-sgk** and abundant **h-sgk** positive inflammatory cells appeared in the lamina propria. Combined **h-sgk** in situ hybridization and immunohistochemical analysis of CD68 antigen expression identified a part of these cells as macrophages. In addition to spatial correlation of transforming growth factor (TGF)-beta1 protein and **h-sgk** mRNA expression, **h-sgk** transcription in human Intestine 407 and HepG2 cells as well as in U937 monocytes/macrophages was strongly induced by TGF-beta1 in vitro. CONCLUSIONS: **h-sgk** expression in normal and inflamed intestinal mucosa may be regulated by TGF-beta1 and may contribute to the pleiotropic actions of TGF-beta1 in mucosal cell populations.

L16 ANSWER 12 OF 15 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
ACCESSION NUMBER: 1998-10366 BIOTECHDS
TITLE: New nucleic acid encoding cell-volume regulating kinase

h-sgk and related proteins;
enzyme and protein used for diagnosis and therapy of condition related to cell-volume change

AUTHOR: **Lang F; Waldegger S**
PATENT ASSIGNEE: Dade-Behring-Marburg
LOCATION: Marburg, Germany.
PATENT INFO: EP 861896 2 Sep 1998
APPLICATION INFO: EP 1998-101338 27 Jan 1998
PRIORITY INFO: DE 1997-1008173 28 Feb 1997
DOCUMENT TYPE: Patent
LANGUAGE: German
OTHER SOURCE: WPI: 1998-449109 [39]

AB A nucleic acid (A) that encodes the human cell-volume regulating serum and glucocorticoid-dependent kinase (**h-sgk**) with a given 431 amino acid protein sequence is claimed. (A) has a given 2,370 bp nucleotide sequence. Also claimed are nucleic acids that hybridize with (A) under stringent conditions and encode an active cell-volume regulating kinase, the transcription of which is not induced by fetal cattle-serum or glucocorticoids. Alternatively it can encode a kinase that is not identical with rat-sgk. The claims also cover polynucleotide fragments consisting of bases 980-1,480 of the given sequence that encodes an immunogenic fragment of **h-sgk**. The claims extend to recombinant **h-sgk**, and receptors that specifically bind to **h-sgk**. The new nucleic acids are used to detect (A) by Northern blotting and hybridization. The protein **h-sgk** can be used to detect receptors which can be used to detect and quantify **h-sgk** in immunoassays. This has application in diagnosis and therapy of conditions associated with cell-volume changes, including hyper-and hypo-natriemia, diabetes mellitus, fructose intolerance, Alzheimer disease, etc. (15pp)

L16 ANSWER 13 OF 15 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN DUPLICATE 5

ACCESSION NUMBER: 1998305122 EMBASE
TITLE: Cloning of sgk serine-threonine protein **kinase** from shark rectal gland - A gene induced by hypertonicity and secretagogues.
AUTHOR: **Waldegger S.**; Barth P.; Forrest J.N. Jr.; Greger R.; **Lang F.**
CORPORATE SOURCE: S. Waldegger, Department of Physiology 1, University of Tubingen, Gmelinstr. 5, D-72076 Tubingen, Germany
SOURCE: Pflugers Archiv European Journal of Physiology, (1998) 436/4 (575-580).
Refs: 35
ISSN: 0031-6768 CODEN: PFLABK
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Recently, the cell-**volume-regulated** serine-threonine protein **kinase h- sgk** was cloned from a **human** hepatoma cell line. The sgk gene was shown to be induced by cell shrinkage in many different mammalian cell lines. In this study, two highly conserved serine-threonine protein **kinases**, sgk-1 and sgk- 2, were cloned from rectal gland tissue of the spiny dogfish (Squalus acanthias). Both **kinases** showed a distinct pattern of tissue specificity, with high expression levels in kidney, intestine, liver and heart. In rectal gland slices sgk-1 transcription was induced by exposure to hypertonic solution, reduction of the extracellular urea concentration, and addition of the secretagogues vasoactive intestinal polypeptide (VIP) and carbachol. The shark sgk-1 serine-threonine protein **kinase**

may therefore provide a link between cell volume, Cl-secretion and protein phosphorylation state in shark rectal gland cells.

L16 ANSWER 14 OF 15 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 97272242 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9114008
TITLE: Cloning and characterization of a putative human serine/threonine protein kinase transcriptionally modified during anisotonic and isotonic alterations of cell volume.
AUTHOR: **Waldegger S**; Barth P; Raber G; **Lang F**
CORPORATE SOURCE: Physiologisches Institut I der Eberhard-Karls-Universitat, D-72076 Tubingen, Germany.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1997 Apr 29) 94 (9) 4440-5. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-Y10032
ENTRY MONTH: 199705
ENTRY DATE: Entered STN: 19970609
Last Updated on STN: 20020420
Entered Medline: 19970527

AB Hepatic metabolism and gene expression are among other regulatory mechanisms controlled by the cellular hydration state, which changes rapidly in response to anisotonicity, concentrative substrate uptake, oxidative stress, and under the influence of hormones such as insulin and glucagon. Differential screening for cell volume sensitive transcripts in a human hepatoma cell line revealed a gene for a putative serine/threonine kinase, **h-sgk**, which has 98% sequence identity to a serum- and glucocorticoid regulated kinase, sgk, cloned from a rat mammary tumor cell line. **h-sgk** transcript levels were strongly altered during anisotonic and isotonic cell volume changes. Within 30 min **h-sgk** RNA was, independent of de novo protein synthesis, induced upon cell shrinkage and, due to a complete stop in **h-sgk** transcription, reduced upon cell swelling. Comparable changes of sgk transcript levels were observed in a renal epithelial cell line. **h-sgk** mRNA was detected in all human tissues tested, with the highest levels in pancreas, liver, and heart. The putative serine/threonine protein kinase **h-sgk** may provide a functional link between the cellular hydration state and metabolic control.

L16 ANSWER 15 OF 15 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 97:351584 SCISEARCH
THE GENUINE ARTICLE: WV421
TITLE: **h-sgk**, a novel human serine threonine protein kinase, is transcriptionally controlled by cell volume
AUTHOR: **Waldegger S (Reprint)**; Raber G; Sailer E; Barth P; **Lang F**
CORPORATE SOURCE: UNIV TUBINGEN, D-72076 TUBINGEN, GERMANY
COUNTRY OF AUTHOR: GERMANY
SOURCE: PFLUGERS ARCHIV-EUROPEAN JOURNAL OF PHYSIOLOGY, (NOV-DEC 1997) Vol. 433, No. 6, Supp. [S], pp. P357-P357. Publisher: SPRINGER VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010.
ISSN: 0031-6768.
DOCUMENT TYPE: Conference; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 0

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=> d his

     (FILE 'HOME' ENTERED AT 09:30:06 ON 28 MAY 2004)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS'
     ENTERED AT 09:30:37 ON 28 MAY 2004
L1           881 S VOLUME(A)REGULATED
L2           157 S L1 AND KINASE?
L3            49 S HUMAN AND L2
L4            19 DUP REM L3 (30 DUPLICATES REMOVED)
L5            39 S "H-SGK"
L6            20 DUP REM L5 (19 DUPLICATES REMOVED)
L7            20 S L6 AND KINASE?
L8       6092312 S CLON? OR EXPRESS? OR RECOMBINANT
L9            73 S L3 OR L5
L10           49 S L9 AND L8
L11           22 DUP REM L10 (27 DUPLICATES REMOVED)
                 E LANG F/AU
L12         3108 S E3
                 E WALDEGGER S/AU
L13          348 S E3
L14         3155 S L12 OR L13
L15           30 S L9 AND L14
L16           15 DUP REM L15 (15 DUPLICATES REMOVED)
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	Issue Date	Pages	Document ID	Title
1	20040304	397	US 20040043930 A1	Novel proteins and nucleic acids encoding same
2	20030102	20	US 20030003559 A1	Cell volume-regulated human kinase h-sgk
3	20011004	15	US 20010027184 A1	Serine/threonine protein kinase (H-SGK2)
4	20011204	19	US 6326181 B1	Cell volume-regulated human kinase h-sgk

	Issue Date	Pages	Document ID	Title
1	20040304	397	US 20040043930 A1	Novel proteins and nucleic acids encoding same
2	20040226	9	US 20040038882 A1	Sgk2 and sgk3 used as diagnostic and therapeutic targets
3	20030102	20	US 20030003559 A1	Cell volume-regulated human kinase h-sgk
4	20011004	15	US 20010027184 A1	Serine/threonine protein kinase (H-SGK2)
5	20011204	19	US 6326181 B1	Cell volume-regulated human kinase h-sgk

	Issue Date	Pages	Document ID	Title
1	20040304	397	US 20040043930 A1	Novel proteins and nucleic acids encoding same
2	20040226	9	US 20040038882 A1	Sgk2 and sgk3 used as diagnostic and therapeutic targets
3	20040212	50	US 20040029857 A1	Heterocyclic inhibitors of ERK2 and uses thereof
4	20030306	159	US 20030044845 A1	Novel therapeutic agents for membrane transporters
5	20030102	20	US 20030003559 A1	Cell volume-regulated human kinase h-sgk
6	20011004	15	US 20010027184 A1	Serine/threonine protein kinase (H-SGK2)
7	20030624	243	US 6583275 B1	Nucleic acid sequences and expression system relating to Enterococcus faecium for diagnostics and therapeutics
8	20011204	19	US 6326181 B1	Cell volume-regulated human kinase h-sgk

	L #	Hits	Search Text
1	L1	4	"h-sgk"
2	L2	225	volume adj regulated
3	L3	6	l2 same kinase$2
4	L4	3	l3 same human
5	L5	19131	LANG WALDEGGER
6	L7	5	l5 and l6
7	L6	8	l1 or l3